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THE THIRTY-SECOND MAUDSLEY LECTURE: SENSORY EXPERIENCE AND BRAIN STRUCTURE*

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It was in my early youth that I first read Bergson's *Creative Evolution*. At that time, as I recollect, it particularly captured my interest and attention because it seemed to embody certain essential principles which I had not found to be expressed quite so forcibly elsewhere. It is true that the validity of many of Bergson's speculations and ideas have since been seriously controverted and so find little or no support today, but his philosophy also propounds certain conceptions which are perhaps worthy of renewed attention. I refer more especially to Bergson's contrast of those two aspects of conscious experience which he terms intellect and intuition. Intellect, he argues, is the product of a gradual evolutionary process which enables the individual with increasing efficiency to select and abstract just those several features of surrounding objects which are directly relevant to the problem of evolutionary survival. In so far as it selects and abstracts, the intellect by itself can provide only a partial view of external reality. "To conquer matter," Bergson says, "consciousness has had to exhaust the best part of its power"—it has had to "adapt itself to the habits of matter and concentrate all its attention on them, in fact, determine itself more especially as intellect." But there remains "around our conceptual and logical thought a vague nebulosity, made of the very substance out of which has been formed the luminous nucleus which we call the intellect". Therein reside certain powers that are complementary to the understanding—powers of intuitive recognition. Intellect is essentially based on a sort of derived symbolism gradually elaborated in the course of evolution, which serves its immediate purpose as a convenient, useful, and indeed essential device for gaining control over the material world; on the other hand intuition, according to Bergson, is capable of giving scintillating flashes of insight "into the very inwardness of life".

It would perhaps be presumptuous for an anatomist to express an opinion on the validity of Bergson's arguments, and I readily confess that I find some of them exceedingly difficult to follow. But it has always seemed to me a peculiarly interesting circumstance that Bergson's conception of a duality of

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conscious experience has some rather suggestive correlates in certain aspects of purely neurological phenomena. The fact is that the idea of a duality in modes of sensory perception is ever and again presenting itself in one form or another to the neurological investigator; indeed, the late Sir John Parsons, in his *Introduction to the Theory of Perception*, went so far as to suggest that "the whole nervous system, receptor and effector, is built upon a dual—dyscritic and epicritic—basis". Head's well-known hypothesis of a dual mechanism to explain his categories of protopathic and epicritic sensation commanded assent for many years, and even though the explanatory postulates which he himself put forward have now proved untenable (at any rate so far as the peripheral mechanism is concerned), the contrasting sensory phenomena which he described as characteristic of the earlier and later stages of nerve regeneration still require to be explained on a neurological basis of some sort. Dual mechanisms in the apparatus of the special senses have suggested interesting parallels; for example, the contrasting rod and cone mechanisms in the retina, or in the apparatus of smell the puzzling association (in most mammals) of a highly differentiated olfactory bulb with the cruder and more primitive organization of an accessory olfactory bulb. In the central nervous system, dual sensory pathways have long been known to exist, but they have gained increasing recognition in recent years as the result of the intensive studies of the so-called "reticular system" of the brain-stem and thalamus. And now, with the discovery and demarcation of secondary sensory areas in the cerebral cortex (somatic, visual and auditory), it seems that duality is a general principle even of cortical organization. I do not, of course, want to insist that this duality of the neural organization of sensory pathways is actually the structural basis of Bergson's contrast between intellect and intuition, or, indeed, that it has any direct relation to it. But there seem to me at least to be some rather interesting parallels which, even if they are no more than distantly analogical, are worth discussion.

About thirty years ago, the American anatomist G. E. Coghill carried out his classical experiments on the anatomy of behaviour in the larval amphibian *Amblystoma*, and showed that the first responses to external stimuli which appear in the course of the development of behavioural reactions are total responses—that is to say, the body and limbs act as a whole. Only later do separate limb movements become individuated as simple and isolated reactions independent of total movements of the body. The total responses are related to a stage in the development of the central nervous system when the latter is composed of a diffuse network of nerve cells and fibres, and the appearance of local, isolated, responses is presumed to be dependent on the "crystallizing out" from the initially diffuse network of circumscribed and segregated tracts of nerve fibres. These experiments were the first to indicate such a sequence of behavioural development, in contradistinction to the rather widely held view that it is the local reflex that is the primary unit of behaviour, and that these reflexes become only secondarily linked up by some integrating mechanism at a higher functional level of the nervous system. Coghill's general conclusions (which were later substantiated by the observations of Barcroft and Barron (1939) on the development of responses in sheep embryos) suggest a broad functional division of types of neural organization into (1) the fundamentally primitive foundation of diffuse networks of cells and fibres which mediate generalized motor and sensory activities, and (2) a more differentiated system of localized cell clusters and connecting fibre tracts which mediate activities of a more specific and circumscribed nature. One of the most interesting points

which I want to stress here is that the primitive foundation still persists in large measure in the adult brain—even the human brain; in fact, it is this which comprises the basis of the whole reticular system.

In a sense, the reticular system of the brain is not a new discovery of recent years—it has simply been rediscovered. For, since the days of the early neuro-histologists such as Cajal, Van Gehuchten, Kölliker and so forth, we have always known of its existence, and we have always known that, besides following the long and more direct ascending pathways to the higher levels of the brain, sensory impulses are also diverted by innumerable collateral branches of entering sensory fibres (as well as by many collaterals of the direct ascending tracts of fibres) into diffuse collections of cells which extend as a sort of continuous matrix all the way up to the thalamus in the forebrain. It has for a long time been apparent, therefore, that incoming sensory impulses may be conveyed to higher functional centres of the central nervous system by two routes—(1) by discrete and well-defined relay stations, and thence to the highest functional levels of the cerebral cortex, and (2) by the diffuse and ill-defined network of the reticular system through which they may be dispersed in almost every possible direction, and eventually conveyed to higher levels largely by a sequence of short inter-nuncial neurons.

The idea presents itself that, just as anatomically the reticular system appears to provide a generalized sort of diffuse matrix or background against which the more specific tracts and nuclei stand out as circumscribed and discrete formations, so it provides the medium for a sort of background of generalized and diffuse sensory activity upon which, so to speak, the main (and immediately relevant) elements of sensory perception are high-lighted as discrete items of conscious experience. At any rate, there is the neural basis for such a functional collaboration, and it fits in rather well with some of the psychological interpretations of sensory experience. But I will return later to a brief consideration of some of the possible sensory roles of the reticular system, because I would like in this lecture mainly to give attention to the manner in which the specific sensory pathways become elaborated to serve their discriminatory functions. Probably it will be agreed that the extent to which we can gain an intellectual appreciation and understanding of the details of our material environment depends initially on (1) the capacity of the sensory mechanisms of the central nervous system for selecting, segregating and analysing the effects of the considerable variety of external stimuli which impinge on our sensory receptors, and (2) on the ability of the higher centres to re-synthesize impulses generated by diverse stimuli with such precision that the patterns which gave rise to them are accurately reproduced in their original configuration. In the language of more modern fashion, these two aspects of sensory perception are sometimes spoken of as “coding” and “decoding”, a terminology which has the disadvantage (in common with the application of a number of other cybernetic terms to biological processes) of begging very vital questions. However, it seems clear that it is the capacity of the nervous system for selecting and segregating the effects of different stimuli which is primarily the most important factor in the development of powers of sensory discrimination, and what seems to be of outstanding interest (though it does not seem to be generally recognized) is that in the course of evolution the progressive improvement of discriminatory powers has to a large extent been determined not (as far as we have evidence) by any increasing elaboration and differentiation of the receptor organs themselves, but by the increasing elaboration and differentiation of the sensory centres which make possible a more complete analysis of gradations of stimuli

to which the receptors themselves are *already* differentially susceptible. Let me illustrate this thesis in the first instance by reference to the visual system.

It is a somewhat remarkable fact that, in general, the intrinsic structure of the retinal epithelium shows no fundamental difference if the human eye is compared with that of some of the lowliest of placental mammals today. Yet the range of visual discrimination in man has reached a very high degree of perfection. This is no doubt partly due to the acquisition of full binocular vision, but the latter has been made possible by a differentiation of the main lower visual centre, the lateral geniculate nucleus, which has so re-arranged its pattern of cells that impulses from corresponding points in the two retinae are conveyed separately to adjacent zones and thence projected on to the same cortical locus. In sub-mammalian vertebrates the optic tracts are usually completely crossed, and in loer w mammals in which only a small proportion of fibres remain uncrossed there is considerable overlap in the geniculate nucleus of the termination of the crossed and uncrossed fibres. But in the human geniculate nucleus, as also in that of the higher Primates, there are separate layers of cells completely segregated from each other in relation to which homolateral and heterolateral retinal fibres terminate. So far as is known, there is no opportunity for functional interaction between these layers in the Primates, and thus no fusion of visual images from the two eyes can occur until the cortical level is reached. It seems evident that the segregation of crossed and uncrossed retinal fibres in each geniculate nucleus is the pre-requisite for full binocular and stereoscopic vision, and it is interesting to consider how this segregation has been achieved. After the decussation of the optic nerves at the optic chiasma, the two sets of fibres become more or less diffusely intermingled, and in each optic tract as far back as the geniculate nucleus those derived from corresponding sectors of the two retinae appear to remain almost entirely mixed up in a random disarrangement. But as soon as they actually enter the geniculate nucleus they become completely sorted out again so that each set is conveyed to sharply separate cell layers. Thus it is not that the laminar disposition of cells in the geniculate nucleus is secondarily imposed on the latter by a primary geometrical arrangement of crossed and uncrossed fibres as they arrive in the optic tracts—rather it is that the laminar differentiation of the geniculate nucleus, by some sort of positive selectivity, *secondarily* sorts out the incoming fibres from each of the two retinae. This phenomenon serves to bring into prominence a major feature of lower sensory centres in general—they are not merely relay stations, as so often seems to be supposed; their main function is that of sorting stations, a function which involves an unshuffling (rather than a reshuffling) of incoming impulses. It is at this level that sensory fibres become sorted out and classified in accordance with the category of impulse which they carry, and it is here that the analytical mechanisms are developed which provide the essential basis for sensory discrimination.

The developmental process whereby the different cell layers of the geniculate nucleus selectively determine the segregation of crossed and uncrossed optic fibres poses one of the most difficult questions of morphogenesis. But such a process of segregation has actually been studied experimentally in lower vertebrates. I refer to the work of R. W. Sperry in which he cut the optic tract in newts and studied the course of regeneration. In his experiments he found that at the site of the lesion the regenerating fibres became tangled in the scar in a quite irregular ravelling, so that they entered the central stump in random disarrangement. But on reaching the optic lobes they were in some manner sorted out again so that fibres from different sectors of the retina established

their proper functional connections with their normal projection areas in the optic lobes. This "selective patterning of synaptic connections" (as Sperry has described it) has so far defied satisfactory explanation. But it has been abundantly demonstrated to occur in the developmental stages of lower vertebrates, and it may be supposed that some such process has perhaps also been involved in the evolutionary differentiation of the neural basis of sensory discrimination in general.

The sorting and sifting of retinal impulses by the geniculate nucleus in man goes much farther than the segregation of crossed and uncrossed impulses, for the fibres from the central area of each retina, and from every locus in the central area, are sorted out into three groups which terminate severally in three sharply-defined cell layers. In other words, the central area of the retina, in its projection on the geniculate body, is replicated three times.* What can be the significance of this triple map of the retina in the brain? When I first drew attention to this arrangement as the result of experiments carried out many years ago, I suggested that it might be related to the trichromatic theory of colour vision, for these experiments provided the first concrete evidence for the existence in the optic tract of a three-fibre unit such as had been postulated long ago on the basis of this theory. I drew attention to certain facts which seemed to support such a proposition, but at the same time emphasized its tentative nature. It is still no more than a provisional hypothesis, and is likely to remain so until it has been found technically feasible to compare by electro-physiological methods the relative spectral sensitivity of the three cell-layers related to each eye. But whatever may be the differential nature of the impulses (or the information conveyed by them) which are segregated at the geniculate level into three categories, clearly a segregation does occur, and it may be presumed to relate to a functional analysis of some sort. Obviously, also, colour discrimination must ultimately depend on a segregation of retinal impulses related to differentially sensitive receptors (though perhaps not in the simple form which I first envisaged in my own interpretation of the trilaminar organization of the geniculate nucleus).

Now, the interesting point has emerged from comparative and electro-physiological studies (particularly those of Granit) that a differential spectral sensitivity of the receptors may exist in lower mammals which appear from appropriate tests to be colour blind; in other words, the structural mechanism which could permit a *peripheral* analysis of the spectrum may be developed in the absence of a sorting mechanism in the brain whereby impulses related to different local parts of the spectrum can be sorted out. It might be difficult to understand such a situation if the spectral sensitivity of retinal elements is presumed to depend on a variety of pigments having different absorption curves, which have (so to speak) become specifically elaborated in direct response to the requirements of colour vision. But my colleague Dr. Barer has drawn attention to another possible explanation of the differential sensitivity—that it depends fundamentally on random slight variations in cone geometry, which determine corresponding differences in the refraction and dispersion of rays of different wave-length as they pass along cones of slightly different angles. "We have here", he says, "a possible filter mechanism which might result in differences in spectral sensitivity among the cone population", some cones trapping red, green, and blue light, others mainly green and blue, and still others blue only. If this attractive hypothesis should prove to be well

* It should be noted that, so far as separate cell laminae are concerned, the more peripheral areas of the retina are only replicated twice, and the extreme periphery not at all.

founded, it is to the highest degree unlikely that there is anything but a continuous gradation in the geometry—and therefore in the “colour-trapping” ability—of the cones. In other words the selection, sorting out, and separation of the three main categories of retinal impulse which (on the trichromatic theory) are required as a fundamental basis for colour discrimination must be determined in the visual centres of the brain. Such an interpretation would conform with my general thesis that the progressive evolutionary development and refinement of powers of sensory discrimination are predominantly effected by a progressive elaboration of the central nervous apparatus which makes it possible to exploit more and more fully the potentialities of receptor mechanisms already in existence. Incidentally, this phenomenon seems to be an excellent example of what evolutionists have termed “pre-adaptation”, that is to say, the incidental development of structural characteristics which are not *directly* adaptive in an ancestral type, but which do become adaptive in its evolutionary descendants.

A similar phenomenon appears to be exemplified in cutaneous sensation. It was for many years supposed that the enhancement of cutaneous sensory discrimination in higher types of organism depends on the progressive differentiation in the latter of various kinds of specialized receptor, e.g. Meissner's corpuscles for touch, Krause's end-bulbs for cold, Ruffini terminals for warmth, and so forth. However, it has now been demonstrated (particularly by Weddell and his collaborators) that these elaborations of nerve endings in the skin, whatever their real significance may be, are by no means an essential basis for the finer grades of sensory discrimination, for our discriminatory powers are quite well developed in areas of skin which are simply innervated by a diffuse network of free nerve-endings. But such an arrangement of interlaced free nerve-endings appears to be common to the whole range of vertebrates; in other words, it seems that, anatomically, the peripheral mechanism of cutaneous sensation remains more or less stationary throughout the course of vertebrate evolution. Like the visual system, however, the potentialities of this peripheral mechanism have been exploited by sorting devices which become primarily differentiated in the central nervous system. There is good evidence, both direct and indirect, of the existence of these devices. For example, when they reach the brain or spinal cord, cutaneous nerve fibres become to a considerable extent re-grouped both in respect of their topographical distribution in the skin, and in accordance with the functional category of impulse which they convey. Thus, fibres coming from diverse areas of the skin become thoroughly mixed up in random fashion as they travel back centrally in the main nerve trunks. But between the posterior root ganglion and the spinal cord, by some mysterious process they become once more resorted into fasciculi each of which is made up of fibres innervating a local area of the skin. Clearly this is a device which makes possible tactile localization by segregating from each other the actual terminations of impulses received from different local areas. It has likewise been demonstrated that there is a resorting of impulses related to different sensory modalities, for different rootlets of each spinal nerve may be composed of different categories of fibres—one rootlet may convey fibres subserving superficial tactile sensation, and an adjacent rootlet may convey only those subserving deep or proprioceptive sensibility (Kuhn). There is further evidence that fibres which are predominantly concerned with the transmission of “pain impulses” become segregated out in the posterior roots, and the well-established fact that impulses related to different modalities are to a large extent segregated in different ascending tracts in the spinal cord and brain stem is also difficult of

explanation except on the basis of a selective sorting of sensory fibres when they reach the central nervous system.

As with the visual system it seems evident that the re-grouping of somatic sensory fibres (and of the impulses conveyed by them) as they enter the nervous system must be determined by some sort of positive selectivity exerted by the different groups of receiving cells in the lower sensory centres—that is to say, the analysis of the sensory inflow must depend primarily on their activity. We are entirely ignorant of the actual process underlying such a selection of cutaneous impulses, but, again, there is experimental evidence that it does occur. For example, it has been found that if in tadpoles a strip of the belly skin is transplanted to the back, it becomes innervated abnormally by the dorsal (instead of the ventral) rami of the spinal nerves. Yet when, in the mature frog, the transplanted skin on its back is touched, the animal wipes its belly with its fore-limb (as though the strip of belly skin were still in its normal position). Similarly, if an extra limb bud is grafted on the back, it becomes innervated by cutaneous fibres destined normally to innervate the trunk skin. But in the adult animal normal cutaneous reflexes were elicited from the limb “indicating that the sensory fibres had formed functional connections in atypical areas of skin and *central relations appropriate for those atypical terminals*” (Sperry). Further experiments of the same kind showed that if a posterior spinal root of one side is transplanted so as to enter the opposite side of the spinal cord, its fibres still make the appropriate connections with the sensory centres so that sensory impulses of different types each elicit the normal responses.

If time permitted, it would be possible to extend these observations by enlarging on the importance of the “sorting out” functions of the lower centres in the sensory pathways. Thus, the fibres from the cochlea of the inner ear terminate in most complicated nuclei in the hind-brain which anatomically can be subdivided into many groups. Indeed, it has been estimated that the length of the basilar membrane of the cochlea is replicated about a dozen times in as many cell groups. “In other words, the cochlea is ‘unrolled’ at the cochlea nucleus, not just once, but repeatedly” (Galambos). We do not know the precise functional significance of this multiple representation in the lower auditory centres, but it can hardly be doubted that (like the multiple maps of the retina in the geniculate nucleus) it is concerned with a sorting and un-shuffling of information derived from the peripheral receptors so as to serve the requirements of a high degree of discrimination. There is also suggestive anatomical and physiological evidence that the several hundreds of discrete glomeruli in the olfactory bulb may provide the structural basis for the sorting and segregation of olfactory impulses.

Naturally, it must be supposed that, once the analysis has been effected by the selective activity of lower sensory centres, the segregation of these impulses is sustained up to the point where they reach the functional levels concerned with their final integration. There is no need here to recount in detail the evidence confirming this suggestion, for it is well known. The segregation of impulses from the retina to which I have already made reference is known to be continued (at least in the higher Primates) until the visual cortex is reached. Auditory impulses derived from different parts of the cochlea retain their spatial sequence through the medial geniculate nucleus up to the auditory cortex, so that in the latter there is rather a precise localization fore and aft in terms of vibration frequency. Recently published studies by Mountcastle, based on single unit analysis, have demonstrated that in the somatic sensory

cortex the vertical cell columns (which are so clearly visible in ordinary microscopical sections) form a sort of mosaic, each vertical column being in most cases "modality-pure" in the sense that it responds only to one type of stimulus (i.e. movement of hairs, pressure on the skin, or mechanical deformation of the deep tissues). And there is also evidence that such a segregation is primarily effected at lower functional levels, e.g. in the posterior column nuclei of the medulla. Without expanding on the general theme of spatial localization in the cerebral cortex, and its well-established relationship to functional localization, it perhaps deserves some emphasis at this time because it seems to me that in recent years quite a deal of unnecessary verbiage has been expended in discursive articles relating to the concept of cortical localization—particularly in reference to cortical architectonics. Let it be fully recognized that the conception of cortical architectonics has a very real basis in the sense that a number of cortical areas are clearly to be distinguished by structural differences, and that the latter are correlated with differences in fibre connections and corresponding functional differences. The fact that some anatomists and physiologists have tended at times to push the conception too far does not invalidate the principle as a whole. Undoubtedly the integrating and synthesizing properties of the cerebral cortex comprise its outstanding functional significance, but integration is not possible unless there is something to integrate—analysis must precede synthesis. It follows that the cortex must first of all be presented with all the elementary components of sensory perception before it can weave them into a coherent whole, and it is clear that these elementary components are represented by a diversity of impulses initially sorted out by the lower sensory centres and projected to the cortex in more or less well-defined spatial and temporal patterns. Some of the anatomical correlates of the spatial patterns are to be seen in the diversities of cortical structure.

The proposition that the lower sensory centres of the central nervous system function as sorting stations involves a number of considerations. One of these is that, in the process of sorting incoming impulses, they appear to select and classify them in certain fundamental categories. It seems to me rather important to recognize this function. Some of the sensory physiologists of the past have been criticized for their attempts to codify cutaneous sensations in terms of sharply defined categories such as those of touch, warmth, cold and pain, and to relate them to different morphological types of sense-organ in the skin. Such criticisms have some justification so far as they apply to the supposed peripheral anatomical basis of cutaneous sensory discrimination, for (as already noted) diffuse, interlacing networks of free nerve endings with no obvious morphological differentiation seem to be adequate for quite a high degree of discrimination. Clearly, however, there must be *some* sort of a preferential sensitivity in different nerve endings, or in varying local patterns of nerve endings, in relation to different types of stimulus. If it is one of the main functions of lower sensory centres to sort, they must have a range of material from the periphery which is capable of being sorted. But it now seems more likely, on anatomical and physiological grounds, that it is not the sensory endings in the skin that are themselves divided into quite sharply defined categories of differential sensitivity—rather they may represent in their range of sensitivity a more or less smooth continuum with no very abrupt demarcations between those which are preferentially responsive to different stretches in the total range of stimulation. One functional type of cutaneous receptor may tend to grade almost insensibly into the next (at any rate to the extent that receptors of differential sensitivity are more closely graded than is commonly reckoned

to be the case). In other words, the apparent sharpness of contrast in the cutaneous sensory modalities which we are accustomed to distinguish subjectively may depend not so much on an equivalent abruptness of contrast in the peripheral receptors themselves, as on the selection and classification, by the sorting action of the specific sensory centres in the nervous system, of impulses related to certain restricted foci in what is otherwise a more or less generalized field of sensory excitation. It is thus primarily in the lower centres that the sensory impulses are codified in the sharply-defined categories which we come to interpret in terms of sharply-defined modalities of sensation. It may also be surmised that impulses initiated by continuous gradations of stimuli affecting special sense receptors in general are likewise classified and sorted into compact groups on their arrival in the brain. By way of analogy, we may refer again to colour discrimination, which appears to operate as if on the basis of selective sensitivity to three local bands of wave-lengths in the red, green and blue parts of the spectrum respectively, and thus might be explained by the selection and segregation in the central nervous system of impulses initiated from these bands.

In addition to the selective action of lower sensory centres on the sensory input, the latter is always susceptible to modification by centrifugal systems of fibres derived from higher functional levels of the nervous system. Although examples of such centrifugal control have been known anatomically for more than half a century, it is only recently that their functional importance has been demonstrated by experimental observations. Centrifugal nerve fibres run forward in the optic tracts to the retina, and from the hind-brain to the sensory cells in the cochlea. The olfactory bulb is concerned with receiving and sorting impulses conveyed from the sensory cells in the olfactory epithelium, but the resultants of its activity are also controlled by centrifugal fibres running forward from the basal region of the cerebrum. The reactions of cutaneous receptors have been shown to be capable of modification by efferent impulses conveyed through the sympathetic nerve supply of the skin. Many other examples of such centrifugal systems could be enumerated; indeed, it now seems certain that at every level in every sensory pathway, as far up as the cerebral cortex itself, the pattern of input from peripheral receptors may be continually controlled and modified by the activity of central regulative mechanisms. This general principle of nervous organization is obviously of the greatest importance for the study of perceptual phenomena, for it determines that what we ultimately experience as a conscious sensation is not solely dependent on external stimuli alone; the effects of the latter may be variably conditioned by intrinsic activities emanating from the brain itself. In other words, altogether apart from the initial selection and fractionation of sensory impulses in the afferent pathways, there is opportunity for still further selective control of these impulses by efferent pathways. Obviously, therefore the sensory material which ultimately reaches consciousness may be limited and fractionated to a degree which is hardly realized.

If it is the function of the lower centres of the specific sensory pathways to select and sort into categories impulses which have been initiated at the periphery by a continuously graded range of stimuli, if, that is to say, the effect of their selective activity is the abstracting of isolates from a continuum, clearly the higher centres (including the cerebral cortex) are likely to be presented with only an incomplete and patchy replica of external reality by these particular pathways. It is as though we were presented with a series of arbitrary points on a continuous curve, with the result that we interpret the abrupt contrasts between

such discontinuous points as if an even curve did not exist. Such a conclusion makes an interesting comparison with one of the main themes of Bergson's philosophy to which I have already drawn attention—that purely intellectual processes inevitably involve selection and abstraction to the extent that they can only provide a partial view of external reality. I do not wish to push this comparison very far, for I have already emphasized that it may be no more than a very distant analogy. But it is too tempting to refrain altogether from enquiring further whether in the organization of the nervous system there is in fact any counterpart to what he termed “a vague nebulosity around our conceptual and logical thought”, which he supposed to represent intuitive processes.

Now, I have already called attention to the fact that there are two main routes in the central nervous system which are traversed by sensory impulses from the periphery when they reach the spinal cord and brain—(1) the specific pathways which I have so far been discussing and which convey impulses by well-defined fibre tracts to circumscribed sensory nuclei, and thence more or less directly to clearly localized regions of the highest functional levels of the nervous system, and (2) the non-specific or diffuse system, that is to say, the so-called reticular system. The reticular system is exceedingly difficult to define simply because it does not clearly define itself in terms of anatomical topography. It can be visualized as a sort of central core composed of scatterings of nerve cells entangled in an irregular and closely meshed network of nerve fibres, and extending up from the spinal cord through the brain stem to run into continuity with the intralaminar nuclei of the thalamus. As is now well known, the whole system is eventually linked up indirectly with the cerebral cortex and by circuitous routes is capable of influencing and profoundly modifying cortical activity as a whole. Into the reticular formation stretching through the spinal cord and brain stem there stream numerous collaterals from the incoming sensory fibres of peripheral nerves, as well as a continuous succession of collaterals from many (perhaps all) of the ascending tracts of the specific sensory pathways. Thus, on the sensory side of brain function, there is abundant opportunity for impulses initiated by every variety of sensory receptor to activate the reticular system and through the latter to modify fundamental processes of cortical activity. In recent years considerable progress has been made in the anatomical analysis of the reticular system, and one of the important points which has emerged is that it is not quite so diffuse or non-specific as some had supposed. For example, Brodal in the Anatomy Department of Oslo University has demonstrated that in the brain stem many of the cells of the reticular system are disposed in more or less well-defined groups, and some of the latter have rather well-defined fibre connections. Similarly, the intralaminar nuclei of the thalamus have been shown by Powell and Cowan, working in my own department, to have localized and rather precise projections to different regions of the corpus striatum. (Incidentally, this work, taken in conjunction with other studies in recent years, has led to a virtual “rediscovery” of the corpus striatum, in the sense that it is now obvious that this mass of grey matter is far more than the relic of a primitive motor system of the forebrain as commonly supposed; it is in fact a highly important “sensory ganglion” which receives, by way of the intralaminar nuclei, patterns of afferent impulses mediated by the reticular system as a whole.) But although the reticular system certainly shows some intrinsic differentiation of cyto-architecture and fibre connections, it does not appear, on either anatomical or physiological evidence, to provide for the sharp segregation of sensory impulses into different categories such as evidently takes place in the lower centres of the main or specific sensory

pathways. It might be supposed therefore, that the information conveyed through the system to the higher functional levels would be less completely analysed and thus less clear-cut so far as sensory perception is concerned. To this extent, indeed, the information which it transmits to the higher functional levels may actually be a more accurate reproduction of the real nature of things, for the very reason that the natural interrelationships of the initial sensory stimuli are more freely retained, and therefore the information which they provide is presented more as a unitary whole than as a variegated mosaic of discrete sensations. We might say, perhaps, that the specific sensory pathways dissect out essential and immediately relevant items of information from a common matrix of sensory stimuli, while the non-specific pathways convey the impression of the matrix as a whole. These are speculations which naturally arise from the circumstance that for this lecture I have deliberately used certain Bergsonian conceptions as a sort of text to introduce my subject. Pursuing the same theme, I may further recall that Bergson equated "intuition" very closely with "instinct", for he remarks that "by intuition I mean instinct that has become disinterested, self-conscious, capable of reflecting upon its object and of enlarging it indefinitely". It is therefore rather interesting to note, in connection with my speculative correlations, that in birds where activities may be said to be almost all basically instinctive, the higher functional levels of the sensory pathways appear to be represented entirely in the homologies of the intralaminar nuclei of the thalamus (i.e. the thalamic extension of the reticular system) and the elaborate corpus striatum. So far as is known, the cerebral cortex of the avian brain (which in any case is of very trivial extent) receives no ascending pathways directly from the thalamus. If then, as appears certain, instinctive behaviour in lower vertebrates is mediated at the higher functional levels of the brain by the intralaminar nuclei and the corpus striatum, it is to be supposed that the equivalent system in mammals (and Man himself) may be concerned with equivalent functions. At any rate, it seems to me to be a point worth consideration in the light of Bergson's suggestion that intuition is really the ultimate development of instinct.

It must not be supposed that in the mammalian brain the specific and non-specific sensory pathways are quite independent systems. On the contrary, they are closely linked at all functional levels by interconnections. At all levels, therefore, the activities of the reticular system are susceptible to modification by sensory impulses ascending by way of the well-defined tracts of the specific pathways, and these sensory impulses in their turn can be modified by impulses transmitted from the reticular system. The functional interaction between the two systems at the cortical level is still uncertain, and one of the problems now awaiting investigation is the significance of the secondary sensory areas of the cerebral cortex. It is known, on physiological evidence, that the secondary areas receive impulses directly or indirectly from the thalamus, but (unlike the primary areas) it has not so far been possible to trace the source and course of the fibres conveying these impulses. For while the excision of (say) the primary somatic area is followed rapidly by a focal degeneration of cells in the main sensory nucleus of the thalamus, excision of the secondary somatic area alone gives rise to no perceptible change. It is interesting to conjecture the possibility that the secondary sensory areas of the cortex (which, in terms of functional localization, are more diffuse and overlapping than the primary areas) may represent the cortical level of the diffuse sensory pathways of the reticular system of the brain, thus providing a background of more general information against which the individual items, segregated and classified by the

sorting activities of the lower sensory centres, are high-lighted as the "luminous nuclei" of Bergson's analogy. Perhaps, with an effort of mind, one can occasionally get a glimpse of this general background and thus, by a flash of intuitive insight, suddenly recognize relationships and conjunctions between the highlights which had previously been obscure and unobserved. At any rate, it is an intriguing conception, and perhaps quite an apposite one, that sensory impulses poured into the reticular system of the brain may, through its plurality of multisynaptic connections, set going inter-weaving and variegated patterns of activity as it were at random, or at any rate uncontrolled by predetermined selective influences, and that the resultants of this activity may every now and again be fed back into the higher functional levels of the specific sensory pathways, there to be abstracted and construed in terms of intellectual concepts revealing new coherencies and new congruities hitherto unperceived and unexpected. Perhaps, indeed, a mechanism of this sort is the elementary structural basis of what has been termed inventive thinking, that is, imagination. If the criticism should be made that, with our present ignorance of brain-mind relationships, the basis for such a suggestion is too tenuous for it to be considered even as a purely hypothetical idea, it may be answered that of course it is tenuous—but it is at least permissible to suppose (1) that ultimately there must be a neurological correlate of inventive thinking, and (2) that this correlate, however complex it may be, must subsume somewhere within its organization just such interacting systems as I have envisaged.*

Some of my digressions in this lecture are of course frankly speculative. This has been my deliberate intention, partly because I think a lecture of this sort invites the ventilation of ideas rather than the reiteration of facts, and partly because it seems to me that in the present state of neurological enquiry (and more particularly the central problem of brain-mind relationships) there is a very real need for the formulation of new ideas to supplement the routine investigations of the microscope and calculating machine. But let me now briefly refer once more to my main theme (for which a considerable body of evidence has accrued in recent years)—that sensory discrimination is primarily determined by the selective agency of the lower sensory centres of the brain and spinal cord, as the result of which sensory impulses related to different functional categories are unshuffled and sorted, canalized into specific and stereotyped channels, and thence projected in set patterns to the higher centres at a cortical level. In other words, so far as the specific pathways are concerned the sensory input of the central nervous system is very far from being a random input. I said earlier that the fundamental processes underlying this selectivity constitute one of the most difficult problems of the morphogenesis of the central nervous system. It would perhaps be more in accordance with the facts to say that at the moment it has all the appearance of being an insoluble problem. It may be that the interesting analogy of computing machines will help toward an understanding of some of the more elementary manifestations of cerebral activity, but there is one obvious contrast which presents itself. The connections which are required in a machine to permit the sorting and analysis of relevant data are planned and constructed for the machine. The living and developing brain plans and constructs its own connections, and even

* As an extension of this hypothesis, it may be presumed that such interacting systems also underlie one of the most remarkable features of dreaming—that however unwonted or grotesque the juxtaposition of the individual items of a dream may be, they do commonly form a coherent and sequential whole. The oddments of random recollections are, so to speak, strung together by the integrating effects of cortical activity into a consistent story—a story which (as well recognized) can often be shown by analytical methods to be full of meaning.

if after they have been established in the early stages of development these are smashed and pulped to an unrecognizable tangle, it can reconstruct them. Perhaps no contrast is more expressive of the fact that the brain contrives—the machine is only contrived.

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ALCOHOL, ALCOHOLISM AND CONDITIONING: A REVIEW OF THE LITERATURE AND SOME THEORETICAL CONSIDERATIONS

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INTRODUCTION

ALCOHOLISM is a grave social, economic and psychiatric problem which has attracted the attention of countless research workers and theorists. From a survey of the recent literature two conclusions emerge. Firstly, that there is at present no general agreement as to the aetiology, dynamics and treatment of alcoholism (1). Secondly, that the inadequacies of the majority of the experiments reported are such that the findings are often difficult to interpret and highly limited in their value (2, 3). In many cases the conclusions do not follow from the data; in others they are impossible to evaluate because insufficient procedural details are given; other studies attempt to answer a large number of questions and answer none adequately; some findings are based upon extremely small or atypical samples while others are based upon experiments open to such obvious criticisms that serious consideration is unwarranted. Many of these studies are apparently the by-product of the busy clinician's experiences and consequently lacking in rigour of any description. Remarkably few are predictive in their techniques or based upon any clearly formulated and testable theory. It is hardly surprising that so many fail to satisfy the usual criteria of acceptable scientific research (4).

In the present article emphasis will be largely on those studies and techniques relating to conditioning and learning. It is proposed to review the existing literature and to indicate where, in the author's opinion, more research is needed. Whenever appropriate an attempt will be made to provide a rationale within a specific framework of conditioning and personality theory. The relationship between conditioning and personality has been the subject of much recent controversy and it is essential that this issue be clarified before proceeding any further.

It is usual to differentiate between Pavlovian or classical conditioning (in which the only essential criterion is that the conditioned stimulus and the unconditioned, or reinforcing, stimulus are associated by contiguity in some way) and instrumental conditioning (in which the subject receives the reward or avoids the punishment only if he makes the correct, i.e. the conditioned response). Furthermore, classical conditioning may itself be regarded in two ways; if association by contiguity is considered as sufficient then any two stimuli may theoretically become associated with each other so that the occurrence of one evokes the response to the other; if, however, a reinforcement theory of classical conditioning is adopted then the performance of the conditioned response must reduce some learned or acquired drive. It is by no means universally agreed that these categories can be subsumed under the

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heading of conditioning. Thus eminent workers such as Kanorski (5) and Thorpe (6) hold diametrically opposed views.

The many parameters involved in all kinds of conditioning are discussed briefly by Guthrie (7) and in detail by Hilgard and Marquis (8). There is ample evidence to suggest that the apparent ease in formation of classical conditioned responses, the nature of these conditioned responses once formed, and their resistance to extinction depend in part upon variables such as the reflex under investigation, the modality, duration and intensity of the conditioned and unconditioned stimuli, the nature of the temporal overlap between these two stimuli, the intertrial interval, the conditioning procedure used (e.g. partial or 100 per cent. reinforcement) and the method of measuring the conditioning (e.g. amplitude, frequency, latency or resistance to extinction). However, it is not these secondary or external variables that are of primary concern here, for they only partially decide the nature of the conditioned response. According to Pavlov (9, 10, 11) all forms of conditioning are the direct consequences of two cortical processes, *excitation* and *inhibition*, and the nature of the conditioned response is a manifestation of the laws of cortical functioning as well as being dependent upon the external parameters listed above. It is not relevant to speculate about the physiological property of these hypothetical processes; it is sufficient to consider both as positive molar constructs by means of which it is possible to make predictions about personality and conditioning.

If all forms of conditioning are largely dependent upon certain properties of the central nervous system, then the general concept of "conditionability" becomes a meaningful one. It is then possible to speak of a person as being relatively good or bad at forming and retaining conditioned responses irrespective of the reflex used and the technique used to produce and measure the response. If, however, the nature of the conditioned response is largely dependent upon such variables as mood, attention, volition, motivation, conditioning technique and reflex studied, etc., then the concept of general conditionability becomes of little practical value and all predictions must be made in terms of a specific reflex and a specific conditioning situation. This problem has never been adequately investigated; it is unknown whether a general factor of conditionability exists and, if so, how much it contributes to the variance of any particular conditioning data.

There are other problems which limit the applicability of conditioning techniques and which require clarification at some stage. For example, the relationships between the various forms of classical conditioning and instrumental conditioning are unknown and the same problem of a general factor arises. It is also necessary to know more about the relationships between age, sex, intelligence and conditioning, the existing evidence being conflicting (7, 8, 12, 13). Much of the confusion is a result of the multiplicity of techniques used to obtain the data and the diversity of species studied. Bearing in mind the difficulties associated with the concept of conditionability, it is possible from the experiments of the writer to draw several tentative conclusions about conditioning in man. These experiments were carried out under rigorously controlled conditions in a sound-proof conditioning laboratory specially constructed for this purpose (14). The major reflex studied was the conditioned eyeblink to a tone, although in some cases a psychogalvanic skin response was also recorded.

The evidence suggests that:

1. Introverted normals condition better than extraverted normals (15);

2. Introverted neurotics (anxiety states, obsessives, reactive depressives) condition better than extraverted neurotics (hysterics and psychopaths) (12, 16);
3. In both normal and neurotic subjects there is no apparent relationship between conditionability and amount of neuroticism (12, 15, 16);
4. Contrary to predictions based on a Hullian-Spence learning theory, irrelevant drives, such as hunger in an eyelid conditioning situation, apparently have no influence on conditionability (17);
5. Both amylo-barbitone (sodium amytal) and methyl-pentynol (oblivon) (central depressants) make it more difficult to establish conditioned responses and more easy to extinguish them once formed; dexamphetamine (a central stimulant) has the opposite effects (18, 19, 20);
6. In accordance with the Pavlovian concept of excessive excitation setting up a protective inhibition, it has been found that the first effects of a large dose of dexamphetamine tend to be opposite to later effects (19).

In the above studies the personality framework used is that developed by Eysenck (21), in which introversion-extraversion and neuroticism are conceived of as orthogonal dimensions, these dimensions being measured by means of personality questionnaires. It should be noted that in general these results apply equally to both sexes and, within the ranges studied, are dependent neither upon age nor intelligence.

Pavlov originally associated neurasthenia in humans with excessive cortical excitation and hysteria with cortical inhibition, but gave little attention to normal personality types (9). However, since it has also been shown that introverts condition well and extraverts condition poorly, it is possible to discuss the personality patterns and psychopathology of both normals and neurotics in terms of Pavlov's variables. For example, the sufferer from an anxiety state, the obsessive, and (to a lesser extent) the introverted normal, tend to be agitated, hyperactive, tense, reflective, highly sensitive to their environment, over-cautious, hesitant, etc. All these characteristics are consistent with a presumed state of excessive cortical excitation and a readiness to form conditioned responses. Similarly, the hysteric, certain psychopaths and the extraverted normal (to a lesser extent) tend to develop fugues, amnesias, gross conversion symptoms and to be unreliable, impulsive and less responsive and sensitive to their environment. All these characteristics are consistent with a presumed state of excessive cortical inhibition and a relative difficulty in forming conditioned responses (16, 22). In the inhibitory group may also be included certain post-leucotomized and certain brain damaged individuals. There is evidence to show that brain-damaged patients condition poorly (23, 24) and some tentative evidence to suggest that leucotomy may have a similar effect on conditioning. It has been pointed out several times that hysteric and psychopathic reactions are not uncommon following prefrontal leucotomy and certain other forms of brain damage (22, 25) and Kennedy has also stressed the resemblance between these categories, postulating a constitutional similarity between these abnormalities (26). The reported finding that depressant drugs decrease conditionability whereas stimulant drugs increase conditionability may now be viewed in terms of changes in the processes of excitation and inhibition. With this background in mind it is possible to consider the specific problems of alcohol and alcoholism in terms of conditioning and personality.

EXPERIMENTAL STUDIES OF ALCOHOL IN RELATION TO
LEARNING AND CONDITIONING

As far as the present writer is aware* remarkably few experiments have been carried out on the effects of alcohol on any form of conditioning in humans. Gantt (27) found that doses of 0.5 to 1.5 c.c. of alcohol per kg. body weight increased the latency of salivary and motor-conditioned responses in five dogs and that larger doses decreased the intensity of the conditioned responses. Mead (28) studied simple finger-withdrawal conditioning to light and shock in six men under the influence of 30 c.c. of alcohol in a 20 per cent. solution, but his results are inconsistent. Although the present writer has shown that a higher amount of alcohol depresses conditioning in man (20) there is clearly a need for well-designed experiments on the influence of various doses of ethyl alcohol on both the learning of new reflexes in humans and the performance (or extinction) of existing reflexes which have been learned prior to the introduction of the alcohol. Control groups would have to be used and the alcohol administered disguised in some way, such as being given in capsule form or by injection through a stomach tube. It is quite possible, since we have pre-conceived notions of what alcohol should do to us, that suggestion would modify its effects (29). It would also be of interest, both from the point of view of suggestion and direct conditioning, to compare beer and spirits, in varying concentrations. In general it seems that the function of the central nervous system is less affected by beer than by spirits; however, under certain circumstances, the disturbance caused by beer may tend to persist longer (3). In view of the Newmans' recent finding that dexamphetamine and caffeine in ordinary therapeutic doses were ineffective in combating the depressant effects of alcohol (30), it would be of interest and importance to determine just how much of these stimulants are required to restore conditioned reflexes reduced or abolished by alcohol and under what circumstances.

Settlage failed to produce a conditioned response in cats while they were under the influence of sodium amytal (31). When the effects of the drug wore off, the conditioned response was easily evoked with no further training. It is possible that sodium amytal depresses the peripheral performance of conditioned responses as well as or perhaps instead of the central learning of these responses. A similar possibility exists with respect to alcohol and remains to be tested. Pavlov was forced to infer the existence of properties of his hypothetical central processes of excitation and inhibition by observation of the behaviour of peripheral organs such as the salivary glands and skeletal musculature. Settlage's findings indicate the hazards of such an inference. The invention of the EEG now makes it possible to observe central changes more directly during the conditioning of any reflex. The Popovs (32, 33) established a conditioned EEG "after image" to sound in humans, then gave their subjects alcohol. They found that the conditioned "after images" consistently appeared much later and much less frequently, from which they concluded that alcohol has a true central action. This is consistent with the finding that alcohol depresses learning in both animals (34, 35) and man (36). However, although the main effect of alcohol is apparently cortical (37), there is ample evidence to show that, unlike the barbiturates, which appear to have a selective action on specific structures, alcohol is a general depressant acting both peripherally and centrally (38). For example, alcohol impairs maze running efficiency in rats (39, 40, 41, 42, 43, 44)

* Because of language and other difficulties it is probable that a considerable number of communications from Eastern Europe have been overlooked.

although it may be, since the animals were in most cases given intensive doses of alcohol over an extended period, that the impairment in learning is not a temporary effect of the alcohol directly but—as with brain damaged individuals—a result of some permanent damage originated by the alcohol. This is one reason why studies of conditioning in *alcoholics* may be of limited value; it is only by studying the effects of controlled doses of alcohol on normal humans that the properties of alcohol may be discovered. It has been suggested that chronic alcoholism is rarely responsible for felonies or major delinquencies whereas acute alcoholic intoxication precipitates many crimes (45, 46). It may be that alcohol reduces those conditioned responses we term socialization by means of which we have learned to obey the rules of the society in which we live. In addition, alcohol may help by reducing or abolishing those conditioned fear or anxiety responses which prevent many of us from carrying out crimes.

In human instrumental learning alcohol has been shown to have a deleterious effect, e.g. driving skill (47, 48, 49, 50) and Link Trainer performance (51). It depresses sexual responses in dogs (52, 53, 54) and reaction times in dogs and man (35, 53). The early German experiments (55) which purported to demonstrate a decrease in reaction time under certain conditions have since been refuted and the original findings shown to be a consequence of faulty treatment of small sample data (2). The only reputable worker to report that alcohol has a stimulant effect is Masserman (56, 57). He found that alcohol depresses specific tasks in cats, but in small doses may act as a mild stimulant of both cortex and hypothalamus, as shown by the increased responses obtained from direct faradic stimulation of these regions. The work of Santessen (58) also tends to support the possibility that under certain conditions alcohol may not always act as a cerebral depressant. This, if correct, suggests that research on the size of dose in relation to its effects on conditioning and extinction in humans needs to be investigated. The atypical findings of these two workers may be explicable in terms of some concept of positive and negative induction or in terms of Pavlov's complex paradoxical phases (9, 10, 11, 59). However, in the absence of more evidence, any further speculations are premature.

In view of the already discussed relationships between personality and conditioning, any experiments on conditioning under the effects of alcohol should take into account individual differences in personality, particularly in relation to introversion-extraversion. If Shagass' finding is confirmed (60, 61), namely that the sedation threshold of hysterics under sodium amytal is much lower than that of anxiety states, then clear predictions relating this threshold to the effects of alcohol on conditioning may be made. The well-known individual differences in alcohol tolerance of normal subjects (e.g. 62) may be related to all the above effects, particularly to their degree of extraversion and conditionability. This, however, fails to solve the problem of which personality type is more likely to become alcoholic for, as has been often pointed out (63, 64), there is no good evidence in favour of the existence of an alcoholic personality prior to alcoholism. A related problem is that of temporary personality changes while under the influence of alcohol; there are very many studies of the personality of chronic alcoholics, but remarkably few studies of changes produced by experimental intoxication, despite the fact that the latter has all the advantages of controlled laboratory conditions. Another partially resolved problem is that of the permanence of these changes. It was once believed that acquired degeneracy in rats due to alcohol could be inherited (39, 65, 66, 41, 40). However, much of these data are unreliable and of dubious statistical significance and better designed studies have failed to confirm this

belief (67, 68, 69). There would seem to be no evidence of genetic transmission of acquired degeneracy, although it may be that the changes incurred are permanent as far as the individual organism is concerned.

CONDITIONED RESPONSE THERAPY

Much of the treatment of alcoholics lacks a clearly formulated rationale (68, 70, 71) and, with a few notable exceptions such as the work of Voegtlin and his associates (71, 72, 73, 74, 75, 76, 77, 78, 79, 80), this comment is applicable to conditioned aversion therapy even when the therapy purports to be in accord with Pavlovian principles (81, 82, 83). Numerous workers have pointed out the need for rigorous procedures (84, 74, 72, 85) but few have taken this advice. One clinician (86) carefully stresses adherence to the laws governing the acquisition of conditioned responses and then asserts that alcohol should be given *after* the patient feels nauseated. Of the many possible procedures there is no doubt that this form of conditioning (backward conditioning) is the least easy to develop and the most readily extinguished. In addition to such theoretical errors there is a lack of uniformity of procedure and a dearth of technical details in the published reports. It is consequently difficult to evaluate the potentialities of conditioned response therapy and it is hardly surprising that reviewers such as Lévy-Valensi (87) conclude that the value of conditioned response therapy so far is fairly limited. The reports range from long lasting and widespread success (88, 89, 90) through qualified approval (91) to complete rejection (92). Feldman (93) concludes that conditioned response therapies offer no better results than treatment by the more dynamic forms of psychotherapy. Numerous clinicians, usually with no supporting evidence, stress the inadequacies of conditioned response therapy unaccompanied by psychotherapy (94, 95, 96, 97, 84, 98, 99). It is often asserted (100, 101, 102, 103, 104) that training the patient to become abstinent is no cure and Edlin (105) prefers to regard aversion treatment merely as a method of rendering the addict abstinent while psychotherapy and other means of support are being instituted. A frequent conclusion is that conditioning therapy is merely "symptomatic treatment" which fails to eliminate the underlying cause (106, 107). However, such critics merely present acceptable evidence in support of their assertions and ignore the possibility that a symptom may itself be as harmful as the disease (108). Conditioning therapy may help the alcoholic to break his habit of drinking and thus enable him to find more socially acceptable ways of solving his problems. In any case there would seem to be no reason why treatment of both symptoms and the underlying disorder should not proceed along conditioning and learning theory lines. It would also seem reasonable to control the motivational factors by such techniques. If the desire to stop drinking is essential to the success of any kind of therapy then this desire may itself be learned. There is much evidence to suggest that attitudinal factors towards alcohol and alcoholism, including the patient's willingness to seek help, are themselves learned and vary from one culture to another (109, 110).

Apomorphine, and more recently, emetine, are the usual drugs used as the unconditioned stimuli in conditioned aversion therapy. It is sometimes erroneously assumed that the conditioned response established is absolutely specific so that the subject may be in the happy state of having an aversion to spirits, but be able to enjoy the pleasures of drinking wine (111). A related erroneous assumption is that the subject has to be separately conditioned against all alcoholic beverages (112).

Apomorphine conditioning is undoubtedly more time consuming for the experimenter than is antabuse therapy. It has the added disadvantage of requiring rigorous procedural techniques. However, it seems that antabuse presents certain hazards as a therapeutic agent. It may cause acneiform eruptions, allergic dermatitis and urticaria. In some patients it may cause fatigue, tremor, headaches, dizziness, gastro-intestinal disturbances, reduced sexual potency and even death (37, 113, 114, 115, 116). For these reasons it may be preferable to use apomorphine, which is agreed by most workers to produce an aversion directly in accord with the conditioning paradigm. (Should the patient become addicted to apomorphine it is always possible to change to another noxious unconditioned stimulus such as emetine.) There is one notable exception to this general agreement. Dent considers that apomorphine is effective in the treatment of alcoholism because of its action on the medulla cells of the brain in suppressing the need for alcohol. Unfortunately, he has never explained this mechanism in print, except to propound a tentative theory in terms of stimulation of the lower centres of the brain (117). Elsewhere he argues that apomorphine is merely a sedative which reduces the anxiety which is at the root of the addiction (118). Vencovsky believes in using emetine (or apomorphine) to establish a direct conditioned reaction, followed by the continued administration of antabuse to ensure the reappearance of the reaction symptoms whenever drinking is resumed (119). This is perfectly consistent, since in the widest sense antabuse therapy must also be regarded as a form of conditioning in which, after the effect of the drug has worn off, the thought, sight, smell or taste of alcohol presumably sets up a conditioned aversion response.

Although the most usual way of producing a conditioned aversion is by the application of some nausea inducing drug, this is by no means essential. Bachet (120) was able to produce conditioned nausea to the sight or taste of alcohol without using any drugs, and as far back as the last century it had been suggested that alcoholism should be treated by associating a painful stimulus with the taking of the alcohol (121). More recently there has been an accumulation of evidence to show that all kinds of conditioned aversions may be readily produced in both animals and man by the application of electric shock as the unconditioned stimulus (122, 123). Using this technique, Kantorovich (124) was remarkably successful in producing a conditioned aversion not only to the taste of alcohol but also to its smell and sight and even to a photograph of the bottles. There is clearly a need for research into methods other than drugs of producing a conditioned aversion to alcohol.

If drugs are used as unconditioned stimuli, then other problems, totally uninvestigated as far as the present writer is aware, present themselves. For example, apomorphine is supposed to stimulate the so-called "vomiting centre" in the brain. Now although the precise location of the centre is in dispute (125, 126) and hence the specific stimulant action of apomorphine not clear, there is good evidence to suggest that the drug has a cerebral depressant action, especially if large doses are taken. According to Goodman and Gilman (37, p. 1004) even small non-emetic doses of apomorphine may at times be hypnotic. It may be that apomorphine, like sodium amytal (18, 19) and oblivon (20), decreases the ease with which conditioned responses of any description are formed. If this is correct then, although apomorphine is successful in producing the unconditioned response of nausea, it will render the formation of the conditioned aversion responses to alcohol considerably more difficult. Such an experiment, using varying sub-emetic doses, needs to be carried out for various

conditioned responses under laboratory conditions. If it should be found that apomorphine does hinder the formation of conditioned responses then two possibilities arise. Either a suitable drug should be found which produces the required unconditioned response and at the same time has a facilitating effect on the formation of conditioned responses or, prior to each session, the patient could be given a stimulant drug such as caffeine or benzedrine which is known to enhance conditioning. The concept of combining apomorphine with some other drug for various reasons is no new one. For example, Lemere and O'Hollaren (127) suggest that sodium thiopentone (pentothal) should immediately precede any conditioning treatment in order to reduce tension. If sodium pentothal acts as a typical depressant and makes it more difficult for conditioning to take place then this combination of drugs is an unwise one.

The above considerations apply to all forms of conditioned aversion therapy in which drugs are used to produce the unconditioned response. Thus Raymond (128) successfully used this technique to treat a fetishist. Furthermore, if it be conceded that all kinds of psychotherapy may be profitably conceptualized as processes in learning or conditioning (e.g. 129, 130, 131, 132, 133, 134, 135, 136, 137, 138, 139, 140) then the use of stimulant and depressant drugs during therapy may also be considered in terms of this rationale. It might be advisable to combine psychotherapy with a depressant drug when it is desired to extinguish a certain learned pattern of behaviour and to combine psychotherapy with a stimulant drug when new learning is being initiated. A preliminary attempt to make use of similar principles has been reported by Agoston (141).

PREDICTING SUCCESS IN CONDITIONED AVERSION THERAPY

It follows from the discussion presented in the first part of this paper that a prediction of success may possibly be made by giving the alcoholic various laboratory tests of conditioning and questionnaire measures of introversion-extraversion. There is some empirical evidence to support this suggestion. Thus it has often been observed that psychopathy (here regarded as a form of neuroticism appearing only in extraverted individuals and related to poor conditioning) contraindicates success (e.g. 96, 84). However these techniques require experimental validation. Should a direct relationship be demonstrated between laboratory conditioning and responses to conditioned aversion therapy using (say) apomorphine it would seriously weaken the arguments of those who assume (118, 142) that the effectiveness of these aversion techniques is in no way based upon conditioning. It may be observed that a similar exposition might be developed for the prediction of success in any form of psychotherapy.

Before discussing what personality type is more likely to respond successfully to conditioning therapy it may be helpful to formulate two related and often confused problems. These are (a) is there such a thing as an alcoholic personality, once the person has become an alcoholic? and (b) what sort of person is most likely to become an alcoholic, i.e. is there such a thing as a predisposing alcoholic personality?

It has often been concluded that alcoholics tend to be introverted (143, 144); it has also often been concluded that alcoholic groups tend to have a high psychopathic (presumably extraverted) element (145, 167, 147). Others have reported such phenomena as "compulsive" drinkers, presumably introverted (148). Numerous workers have attempted to rate alcoholics in terms of introversion-extraversion, but their findings are of little value since the personality

criteria used were based largely on subjective impressions and unvalidated concepts (149, 150, 151, 152). It is hardly surprising that their findings vary widely as to the ratio of introverts to extraverts. There have been many attempts to classify alcoholics (e.g. 101, 153) but they have all met with little success and there is considerable disagreement as to the personality structure of the alcoholic. In most cases it is impossible to decide whether the obtained personality picture is related to the chronic effects of the alcohol or whether it is an accurate description of the pre-morbid personality of alcoholics (154, 155, 156). It is consequently difficult to evaluate many of these studies (e.g. 145, 157, 158, 147). Hansen and Teilmann (159) examined a group of convicted criminal alcoholics. They split them up into those who were abnormal psychologically and were long term detainees and those who were under short term detention and soon released on parole. They found that the former group tended to come from a materially and emotionally undesirable childhood environment, to have a poorer physique and to have a substantial number of subjects with head injuries among its numbers. Despite therapy, only 13 per cent. of this group improved, whereas 35 per cent. of the other group improved even though untreated. It would be interesting to repeat this investigation predicting success and long-term prognosis for both groups by means of conditioning and personality tests and then to give them both similar forms of treatment. Davies *et al.* (160) used the Strauss and Bacon Stability Scale (161) and other criteria to establish a prognostic profile but their measures are multi-dimensional and consequently difficult to interpret.

Other studies have found no differences between alcoholic groups and normal controls. Thus Vogel (162) found no differences in suggestibility as measured by the body sway test and Sutherland *et al.* (64) found no evidence that their alcoholics had abnormal electroencephalograms. Wittman compared 100 alcoholics with 100 matched normals and failed to find any significant differences with respect to personality or background development (163, 164, 165, 166). The general conclusion seems to be that as yet alcoholic and normal groups have not been shown to differ significantly in personality as indicated by a variety of tests and other measures (167, 168, 64, 84, 169, 170). This conclusion is limited since, with remarkably few exceptions (e.g. 3, 171), the measures of personality used have been subjective or complex in their factorial composition. Although, there is as yet no methodologically sound dimensional system of personality which is widely accepted, it is still possible to use personality tests which have high factor loadings on only one factor in some generally recognized dimensional system. There is a need for research into the effects of alcohol on factorially pure measures of personality. Such research could easily be extended to the effects of the widespread addictions, such as morphine, barbiturates, marihuana and even to the much rarer addictions such as paraldehyde, ether, cocaine and chloroform on measures of personality. The problem of what sort of person is most likely to become an alcoholic is considered in the concluding section of this paper.

ALCOHOL AS A THERAPEUTIC AGENT

In the social sense alcohol is widely used as a therapeutic agent to relieve anxiety and tension. Unlike the barbiturates, alcohol is rarely prescribed for this purpose clinically, even on an empirical basis. Unlike the barbiturates almost nothing has been established concerning the effects of various alcoholic beverages on the formation and retention of conditioned responses, although it

would seem highly probable that the general effect is to depress them. There is, however, some evidence concerning the effects of alcohol on unconditioned responses, especially the sexual ones. It has been established that alcohol raises the threshold of the erectile and ejaculatory reflexes in animals, so that more than normal stimulation is required to arouse these reactions. In large doses the sexual response is completely inhibited (53, 54, 172). Gantt also found that alcohol depresses unconditioned sexual responses far more than any other responses such as the desire for food (52). The widespread popular belief that alcohol is a sexual stimulant seems to be incorrect. The reason for this belief may be that alcohol inhibits already formed conditioned responses, hence reducing or abolishing those learned patterns of socially accepted behaviour which restrict the normal expression or following through of sexual desires. Thus these desires are possibly unaltered but the conditioned restrictions which prevent their implementation are reduced. If, at the same time, unconditioned sexual responses are depressed and a state of near impotence is produced it seems hardly surprising that a conflict situation may be established. There is some experimental support; thus Andreyev (173) found that alcohol given to dogs daily for two weeks resulted in the breaking down of a stable well-established conditioned response pattern and at the same time produced an experimental neurosis. On the other hand, under certain conditions alcohol considerably reduces the behavioural symptoms of an already established experimental neurosis (174). According to Gantt (27) alcohol produces these effects by disturbing the central excitation-inhibition balance. It seems that the result depends both on the conditions of administration of the alcohol and on the personality of the recipients.

The above provides a possible explanation for certain problems resulting from alcoholization in normal individuals and attempts to place on a rational basis such generalizations as that of Seliger and Crawford (175) who conclude that alcohol acts as a depressant on the central nervous system and so enables the underlying forces in the personality to find a more direct expression. It also provides a possible—if at present speculative—rational basis for the alcoholic treatment of certain sexual disorders.* Thus the anxious introverted patient who suffers from *ejaculatio praecox* may have excessive socially conditioned responses greatly reduced by alcohol and at the same time the latency period of ejaculation would be considerably increased. Barbiturates are sometimes prescribed on an empirical basis for the treatment of disorders of a similar nature, but alcohol, given under controlled conditions, may have certain advantages and may be less likely to produce drug dependence or withdrawal symptoms. Certainly the vast majority of people who imbibe alcohol are not alcoholics.

Many problems require investigation, such as the correct dosage and method of administration in relation to the subject's personality. Furthermore, the sexual behaviour pattern and desires of the chronic alcoholic cannot be inferred from a study of the effects of alcohol upon the sexual pattern of normal subjects. Thus Levine (179) found a diminished interest in heterosexual relationships in alcoholics. The possibility of permanent physiological changes in the chronic alcoholic provides yet another variable to be taken into account.

* The consideration of sexual abnormalities in terms of conditioning is not new. Over twenty years ago Meignan discussed sexual impotence in this manner (176) and Max (177) successfully treated a case of homosexual fixation by these means. More recently Salter (178) attempted to explain how masochism may arise as a conditioned response and be treated accordingly, and Raymond successfully applied this method to the treatment of a fetishist (128).

ALCOHOLISM AS A LEARNED RESPONSE

If alcoholism can be successfully treated by a conditioning process then it may be that alcoholism itself is a learned symptom. It is possible to discuss alcoholism as a conditioned response either in terms of a reinforcement theory or otherwise. Many psychologists have emphasized that need reduction is not essential for learning (e.g. 180, 181, 182) and it is reasonable to build up a classical conditioning paradigm based strictly upon association by contiguity. However, the majority of learning theorists adopt some form of reinforcement theory in which the basic assumption is that the learning of an association between a stimulus and a response requires the presence of some sort of reward or reinforcement, even if this be only in the form of a drive reduction. Even if alcoholism is regarded as a serious symptom in a psychiatric disorder it seems advisable to apply a learning theory rationale and to enquire what drive or need the taking of alcohol reduces.

Welch (183) analyses the concept of "needs" at some length and splits them up into two general groups—pain reduction and enjoyment augmentation. Within these groups needs may be physiological or acquired, they may be known to the subject or he may be unaware of them, the reduction may be accompanied immediately by pleasure or followed eventually by pleasure. Wilkins (184) probably has a similar system in mind when he says that theories of alcohol motivation may be divided into psychological and pathophysiological kinds. It is possible to hypothesize numerous needs or drives of various categories which alcohol drinking may conceivably reduce. Thus Royer (185) has shown a relationship between thirst and alcohol drinking, but it would hardly seem widely applicable to postulate thirst as the primary drive which alcohol reduces. For Peabody (186) and Strecker (144) alcohol satisfies the need to avoid reality by screening unsatisfactory external and internal existences. McFarland and Borach (187) believe that there is a relationship between a physiological need for more oxygen and alcoholic intoxication. They found that satisfaction of this need by the inhalation of a suitable oxygen mixture produced a permanent improvement in the alcoholic, presumably because he no longer had to satisfy his need for oxygen by absorbing large quantities of alcohol. Westerfield and Lawrow (188) found that the restriction of food intake in rats results in a marked increase of alcohol consumption, so that it may conceivably be the need for food that alcohol satisfies. However, as with thirst, it would not seem reasonable to postulate a hunger drive as the primary need which motivates people to drink alcohol (especially as Conger (189) found that alcohol does not affect hunger motivating approach responses). According to Lolli (190) alcoholism might be an attempt to satisfy the need for love and tenderness. It could be argued that alcohol reduces the strong oral and narcissistic drives which alcoholics are supposed to possess (191).

The most tenable and frequently proposed drive that alcohol is considered to reduce is anxiety. It has been noted that alcohol is taken especially in times of stress (108, 192) and theories of alcohol drinking as a tension reducing activity have often been proposed (e.g. 193, 194). Horton, in a cross cultural study of certain primitive communities, has shown that there is more drinking in "anxious" societies (195). Certainly it seems true that anxiety in some form, like alcoholism, is universal. Dynamic psychiatrists, e.g. Freud (196), Horney (197), and Sullivan (198) agree that anxiety is encountered to some extent in all communities and in all societies. When Strecker, Peabody and others say that alcohol provides an escape from reality, this may presumably be because reality sets up undesirable tensions or stresses which alcohol reduces.

There are many ways of reducing tension or anxiety such as smoking, masturbating, sexual intercourse, gum chewing, sucking sweets. Specific food addiction (199) or general excessive eating may be learned responses which are reinforced by their anxiety-reducing actions. Thus Ullman (200, 201) found that compulsive and vigorous eating was positively related to tension-provoking situations. It is also known that certain individuals and certain ethnic groups react to stress by eating (202). Ferenczi (203) points out that work itself can be effective in reducing anxiety, and Bird (204) describes the case of an "addictive" worker who reacted to stress by continuous work. Why the alcoholic turns to alcohol in preference to these other tension-reducing activities must remain an open question.

Miller (205) was the first person to show that the decrease in the acquired drive of fear or anxiety can serve as the reinforcing agent in the learning of a habit. More specifically, Conger (189, 206) has provided data which support the hypothesis that the habit of drinking alcohol when rats are placed in a conflict situation is reinforced by the fear-reducing effects of the alcohol. He found that alcohol decreases learned fear-motivating avoidance responses in rats, but does not affect primary hunger-motivating approach responses. Although it seems acceptable that alcohol reduces the learned drive of anxiety, Conger is forced to consider several possibilities. It could be that alcohol tends to reduce the strength of all learned drives but does not affect primary ones; it could also be that the effects of alcohol are specific to certain drives, whether learned or primary. For example, Gantt has provided good evidence that alcohol reduces the primary physiological responses to stimulation of genital areas. It does, however, seem reasonably established that alcohol reduces fear or anxiety. The work of Masserman and his associates with animals confirms this (56, 57, 207, 208). It has also been shown that, in rats, alcohol reduces general activity, which may be a measure of general tension (209, 210). The cross cultural studies of Horton (195) suggest that this conclusion is applicable to man also.*

Conger (212) suggests that conflict itself is tension producing and the work of Liddell and his associates (213) supports this view. According to Dworkin (214) alcohol, as well as other hypnotics, such as amytal, nembutal and hyoscine, considerably reduces the behavioural symptoms of tension resulting during the establishment of an experimental neurosis based on a conflict situation. Thus, whether anxiety is regarded as a learned response and discussed in terms of excessive and widely generalized conditioned responses or as a product of conflict situations, it seems that alcohol reduces this anxiety and so provides the reinforcement which a drive-reducing theory of learning postulates.† Thus the act of drinking to reduce tension becomes a learned habit. By a process of stimulus generalization (in much the same way as anxiety itself is a learned generalized response) drinking alcohol in specific tension-provoking situations becomes generalized to drinking alcohol in most situations, whether tension provoking or not. The habit is maintained by those few situations which provide tension and so the learned pattern becomes reinforced.

* There is experimental evidence to show that other depressants have a similar effect. Thus Bailey and Miller (211) found that the barbiturates abolished conditioned fear responses in cats and Bartholomew, Franks and Marly (20) found that oblivon reduces manifest anxiety in humans.

† If anxiety itself is regarded as a learned response then it is to be expected that alcohol would have the effect of reducing already formed conditioned responses as well as the formation of new ones. As has already been stated, this has never been adequately demonstrated. Since it has been demonstrated that oblivon, amytal and other depressants behave in this manner, it would seem not unlikely that alcohol behaves likewise.

The question now arises as to why some people become addicts almost immediately and others not at all, even though they also drink to relieve tension. A possible, if speculative, answer is that addiction is related to the conditionability and personality of the individual concerned. If it is true that introverted subjects condition more readily than extraverts then the introvert will develop more conditioned anxiety. Therefore there will be more situations where the need to reduce this drive exists; furthermore, fewer reinforcements will be required for the habit to be learned successfully and the generalization to numerous situations and alcoholic beverages would be greater. No experiments have investigated both the conditionability and the personality of alcoholics. Many practical and theoretical difficulties arise, such as the elimination of subjects with brain damage or other physiological changes resulting from the excessive drinking of alcohol. Furthermore, it is logically unwise to assume that the conditionability and personality of the well-established alcoholic is the same as in his pre-morbid state. The existing evidence is inadequate and relates largely to the present personality of the alcoholic. Apart from the finding that Korsakov psychotics condition poorly (24, 215) nothing is known about the conditionability of alcoholics in relation to personality or otherwise. The relationship between alcoholism and conditionability may well be a complex one. Thus in the early stages alcoholism may be positively related to conditionability but, in view of the findings that many chronic alcoholics are brain damaged (216, 217) and that certain brain damaged patients condition poorly, the chronic alcoholic may condition very poorly. The inability to control their drinking that is characteristic of alcoholics may thus be attributable in the early stages to excessive drive and conditioning and in the later stages to an inability to condition.

If the aetiology and treatment of alcoholism is considered in terms of conditioning, several interesting possibilities arise. Is it possible, for example, to produce relief of tension and other pleasurable feelings associated with alcohol by conditioning techniques, so that an originally neutral and innocuous stimulus is just as effective? This might be of benefit in counteracting the physiological withdrawal symptoms of alcoholism during the earlier stages of treatment. Thus Rubenstein (218) successfully treated morphine addiction along these lines. If Wilczkowski's conclusions are correct (he used no control group nor adequate statistical precautions) then it is even possible to condition the physiological effect of alcohol (219). There is much evidence that the sedative effects of sodium amytal can be conditioned (220) and Metalnikov and Chorine (221) succeeded in producing conditioned serological reactions in animals to a social stimulus. Razran (222) criticized their findings on methodological grounds, but a better controlled experiment by Smith and Dalinger (223) produced similar results. Kleitman and Crisler (224) conditioned the retching behaviour originally evoked by morphine to a sound stimulus. Eagle (225) reports that Bykov was able to condition urinary secretions and even leukocytosis.* On the other hand, Gantt, Katzenelbogen and Loucks (226) failed to condition the rise in blood sugar after an adrenalin injection was paired with either a buzzer or the "preparations for the injection". However these authors note that hyperglycaemia can be produced under hypnosis merely by the suggestion "you have drunk a glass of sugared water". Furthermore the behavioural effects of alcohol have been produced by suggestion under hypnotism in the theatre on many occasions. After *animal* experiments in the conditioning of various drug

* Which, as many studies have since confirmed, refutes Pavlov's claim that the cortex is essential to conditioning.

effects, numerous authors have independently come to the conclusion that conditioning can only be achieved if the drug concerned produces experiences "meaningful" to the animal (207, 227, 228, 229). Thus it would have to be the whole configuration of feelings associated with drinking alcoholic beverages which would have to be conditioned. There would seem to be two not incompatible lines of approach. (1) To provide the general pleasurable effects of alcohol, including well-being and the reduction of tension by an innocuous conditioned stimulus. (2) To condition or teach the subject to use other stimuli to reduce his anxiety, stimuli which are less harmful than alcohol, e.g. sweets or gum. A novel possibility in this respect may be that suggested by Richter (230). He found that thyroid extract greatly increased the experimental rats' appetite for sugar, so he attempted to induce a craving for alcohol in this way. To his surprise, just the opposite result was produced, the rats either greatly reduced or completely stopped their intake of alcoholic beverages. Since hyperthyroid patients are very rarely alcoholics Richter thinks that a small daily dose of thyroid extract might stop the craving for alcohol, although he presents no rationale for this belief. It might well be, however, that the thyroid functions as a substitute for alcohol in reducing the patient's needs. This hardly commends itself as an effective substitute, but does suggest the need for an investigation of thyroid function in alcoholics.

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A CLINICAL INVESTIGATION OF CHRONIC SCHIZOPHRENIA

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INTRODUCTION

A DETAILED scheme of classification of schizophrenia has been produced by Professor K. Kleist and elaborated by Professor K. Leonhard (Kleist, 1943, 1949; Kleist *et al.*, 1950, 1951; Kleist and Schwab, 1951; Leonhard, 1936, 1957; Schwab, 1949). The validity of this scheme has been established by other investigators (Schneider, 1955; Schulte-von der Stein, 1955). An English summary of the Kleist-Leonhard scheme has recently been published (Fish, 1957). In 1955 an attempt to classify the chronic schizophrenics in the Female Division of St. Nicholas Hospital, Gosforth, according to the Kleist-Leonhard scheme was begun. By means of reading the case notes, enquiring from the medical and nursing staff and short clinical interviews it was provisionally concluded that there were 243 chronic schizophrenics in the female division with an illness of more than 10 years' duration. These patients were investigated clinically, but by the time the author took up his present appointment only 107 cases had been seen. An attempt was made, however, to investigate approximately the same proportion of patients from each ward of the female division with the exception of the geriatric ward. This series seemed to be a fairly representative sample of the chronic female schizophrenics.

At least one clinical interview along standard lines was given to each patient; frequently two or three interviews. A separate interview was conducted to evaluate thought disorder. The ward sisters were questioned personally about the patients and in three-quarters of the cases they filled in a questionnaire on hallucinations and general behaviour. Quite a number of these patients were known to the author in his day-to-day work as consultant psychiatrist to the female division.

The family history was investigated by sending enquiry forms to all next of kin available and by obtaining information from other mental hospitals where relatives had been in-patients. As many of these patients had been in hospital for years and were no longer visited the family history in many cases was incomplete. The genetic findings will be mentioned briefly in this paper but it is hoped to publish them in full.

All cases except two could be classified according to the Kleist-Leonhard scheme. Table I shows the scheme and the number of cases in this series which were assigned to the different forms.

TABLE I

							Cases	Cases
A. Paranoid Schizophrenias:								
1. Typical Forms:	..	..	..	..	..	..		21
Phantasiophrenia	..	..	..	..	..	..	5	
Progressive confabulosis	..	..	..	..	..	..	2	
Progressive verbal hallucinosis	..	..	..	..	..	..	11	
Progressive somatopsychosis	..	..	..	..	..	..	2	
Progressive autopsychosis	..	..	..	..	..	..	1	
Progressive inspiration psychosis	..	..	..	..	..	..	0	
Progressive influence psychosis	..	..	..	..	..	..	0	
2. Combined forms	..	..	..	..	..	..		18
3. Extensive forms. Progressive reference psychosis	..	..						7
Progressive self reference psychosis							6	
Progressive signification psychosis	..	..	..	..	..	..	0	
Circumscribed delusional psychosis	..	..	..	..	..	..	1	
B. Catatonias:								
1. Typical forms	..	..	..	..	..	..		13
Speech-inactive catatonia	..	..	..	..	..	..	4	
Speech-prompt catatonia	..	..	..	..	..	..	1	
Akinetic catatonia	..	..	..	..	..	..	0	
Parakinetic catatonia	..	..	..	..	..	..	1	
Negativistic catatonia	..	..	..	..	..	..	0	
Prosectic (proskinetic) catatonia	..	..	..	..	..	..	2	
Stereotyped catatonia	..	..	..	..	..	..	5	
2. Combined forms	..	..	..	..	..	..		1
3. Atypical forms	..	..	..	..	..	..		2
Iterative catatonia	..	..	..	..	..	..	2	
4. Unclassifiable	..	..	..	..	..	..		2
C. Confused Schizophrenias:								
1. Typical forms	..	..	..	..	..	..		16
Schizophasia	..	..	..	..	..	..	4	
Incoherent schizophrenia	..	..	..	..	..	..	10	
Paralogical schizophrenia	..	..	..	..	..	..	2	
2. Combined forms	..	..	..	..	..	..		6
3. Atypical forms	..	..	..	..	..	..		5
Atypical shiftlike confused schizophrenia	..	..	..	..	..	..	5	
D. Hebephrenias:								
1. Typical forms	..	..	..	..	..	..		22
Silly hebephrenia	..	..	..	..	..	..	6	
Depressive hebephrenia	..	..	..	..	..	..	4	
Apathetic hebephrenia	..	..	..	..	..	..	4	
Autistic hebephrenia	..	..	..	..	..	..	8	
2. Combined forms	..	..	..	..	..	..		0

THE PARANOID SCHIZOPHRENIAS

Forty-six patients were classified as suffering from Paranoid Schizophrenia. The distribution of these cases among the various subgroups is shown in Table II. It will be noted that no cases of Inspiration and Influence Psychoses were found.

We must first consider the typical subforms. Here five cases of Phantasiophrenia were found. The age of onset in these patients ranged from 25 to 55 years. In four cases the age of onset was over 36 and the average age of onset was 39.8 years. There was a family history of mental illness in only one patient, whose brother had been an in-patient in a mental observation ward, but no details of his illness were available.

TABLE II
Paranoid Schizophrenias

	Cases
(a) Typical Forms:	
Phantasiophrenia	5
Progressive confabulosis	2
Progressive verbal hallucinosis	11
Progressive hypochondriacal hallucinosis	2
Progressive autopsychosis	1
(b) Combined Forms:	
Phantasiophrenia and verbal hallucinosis	5
Progressive verbal and hypochondriacal hallucinosis	7
Expansive autopsychosis and verbal hallucinosis	1
Phantasiophrenia and incoherent schizophrenia	3
Phantasiophrenia and paralogical schizophrenia	1
Expansive autopsychosis and incoherent schizophrenia	1
(c) Atypical Extensive Forms (Progressive Reference Psychosis):	
Self reference psychosis	6
Circumscribed delusional psychosis	1

H.D. is a good example of this subform. She was admitted to Park Prewett Hospital on 26 August, 1938. Her age on admission was 39. The duration of the illness at that time was uncertain, but probably it had only lasted a few months. No family history of mental illness was known. She heard hallucinatory voices and spoke to them continuously. She said that her name was Chifu, she was married to Jesus and she had a "saint child" in Heaven. Later on 1 September, 1938 she said "I am here to command and preserve the peace of the world". In the next few years she is reported as being noisy and restless and rambling in her talk. In 1941 there is a report "She is disorganized in her thought processes, she rambles confusedly concerning the past and mistakes the past with the present. She freely expresses delusional ideas arising from her mental confusion or vice versa." She was transferred to St. Nicholas Hospital on 30 October, 1953. She had fantastic hypochondriacal and grandiose ideas at that time. She said that she had seen her head rolling on the floor, a fireman from Winchester had removed her stomach, last month Lloyd George removed her liver and she had two sons by Immaculate Conception. She had auditory hallucinations but these did not play much part in her illness, she was never observed to hallucinate. This state of affairs continued and when seen by me in 1956 she mistook identities, she had bizarre fantastic delusions which were ill held and variable. She became muddled in the sense that consciousness was not impaired but the content of her speech was completely nonsensical, although the grammatical structure was well preserved. She kept herself clean and tidy and was able to help to some degree with the ward work. She had grandiose ideas which were expressed without much affect and she did not become upset if one laughed at her preposterous claims. She frequently wrote letters full of nonsense which she handed to a visiting doctor or matron. She was cheerful and euphoric. In tests for thought disorder she showed some paralogia and paraphasia.

Two cases of Progressive Confabulosis were identified. The age of onset was 34 and 35 years. One patient had a family history of mental illness, her mother died in St. Nicholas Hospital in 1933. From the case notes available the illness could not be classified with certainty.

The case history of A.G.A. illustrates this subform very well. This patient was originally admitted to St. Nicholas Hospital on 2 September, 1937 when aged 34. Her mother had died in St. Nicholas Hospital in 1933 and from the case notes the illness appears to have been schizophrenia. Her illness had been present for a few weeks before admission. She was then depressed and emotional. She asserted that her husband was not her real husband but appeared to be changed in some way which she could not describe. She said that she heard a noise and thought it was due to a sewer with rats running about in it. She also said that she was frightened to have children in case they turned out to be rats. She improved slowly and was discharged on 18 July, 1938. She was apparently well on discharge and continued so until shortly before her second admission on 22 November, 1940. On this admission she lay with her eyes shut much of the time, but ran towards any door which was being opened. She said that she had been admitted because her birth date had something to do with the War and she thought that the clocks were going backward. She recovered and was discharged on 10 July, 1942. She was re-admitted on 27 July, 1943. This time she complained of an hallucinatory voice which called her a dirty brat, and she asserted that people tried to bewitch her husband and do him harm. Later in March, 1944 she was reported as "rambling aimlessly" and in May, 1944 she was discharged. She was re-admitted on 10 May, 1947 and this is the first time

that confabulation was recorded in the notes. The notes say "She was overtaken by men and the Queen or a woman resembling her in a car near Blanchland and was taken by them into a wood to show her sex to two boys and was left unconscious. These people were the military but said they were Germans and that they were going to kill her." She became less disturbed and was discharged on 23 November, 1947. She was re-admitted on 10 June, 1948 complaining that the neighbours talked about her behind and through the wall of her house and that a bright light shone through the wall so that she had to keep her eyes closed in case the light affected her. She also claimed at this time that she was suffering from anaemia due to a snake bite which she had had two years before. Later that year she complained of snakes being in her insides and that she could hear them snoring. In 1949 she told a doctor "Snakes are enormous things, they want to learn to become doctors so they get into your inside." This statement was made some time after a total hysterectomy for cancer of the cervix. In 1952 she ran away and when asked why she produced a tattered piece of crochet work and said this was a pass out given to her three years previously by her mother, who was at that time dressed in the uniform of a nurse. After this reports of confabulation are frequent in the case notes. Then in 1955 she reported that whilst she was in the Hospital garden she saw a long snake hanging down between her legs with a baby hanging on to it. She showed a mild degree of formal thought disorder. There was some paraphasia and a tendency to carry over material from one test into the next.

Eleven cases of Progressive Verbal Hallucinosi were identified. The age of onset ranged from 21 to 50 years, but was only below 36 years in two cases. The average age of onset was 40 years. It is interesting that two out of these eleven patients were very deaf. It could be argued that deafness in these two cases was a pathoplastic factor.

Two patients in this group had had a pre-frontal leucotomy, in one case followed by frontal lobectomy. This patient improved following this treatment and when seen by me nine years after her lobectomy she was not troubled very much by her hallucinations.

The other patient who had a typical Progressive Verbal Hallucinosi of over twenty years duration did not respond to leucotomy and was treated with reserpine. Before this treatment she complained bitterly about hallucinatory voices and was very querulous. Following reserpine she became more subdued and much quieter and became fit enough for discharge, but while on trial leave she committed suicide by hanging. One wonders whether reserpine may not be contraindicated in this syndrome. Affective disturbance is always present in this subform and these patients are often anxious, depressed and tormented. It is well known that reserpine will produce depression in predisposed individuals and it seems likely that in varieties of schizophrenia where there is anxiety and depression, especially in Progressive Verbal Hallucinosi, reserpine is contraindicated because of the risk of suicide.

A family history of mental illness was found in four cases. Two patients had mothers who committed suicide. One patient had a paternal great-grandmother who died in St. Nicholas Hospital, but the case notes of this relative could not be found. One patient had a maternal grandmother and a maternal aunt who died in St. Nicholas Hospital. From their case notes the probable diagnoses were Senile Dementia and Schizophrenia (probably combined Verbal and Hypochondriacal Hallucinosi) respectively.

The case history of C.F. is given here as an illustrative example of this syndrome. She was certified on 21 November, 1932 and admitted to Winterton Hospital from whence she was transferred to St. Nicholas Hospital on 19 December, 1932. She was 48 years old on admission and the duration of her illness was uncertain. According to the certificate "Patient stated to me that a scar on her foot was due to wireless. That she had wireless in her body for years. She also stated that objects she looked at appeared different at times to what they were and the head female attendant's face looked like china." Shortly after admission she said she was His Majesty's wife and should be on the throne and the Institution belonged to her. For the next four years she heard voices and in particular claimed she heard the voices of her relatives who were kept from seeing her. In 1944 she complained that telephones and radio dictated to her and repeated her thoughts. In 1946 she insisted that the staff kept her visitors away and gave letters addressed to her to patients who impersonated her. In 1947 she was

working well in the hospital kitchen. In 1949 she asserted her husband was not dead as she could hear him and that her daughter was still in Jarrow because she could hear her talking to her. In 1955 she was still expressing the idea that her visitors were kept from her. In 1956 she complained of whispering on the "devils wires". She had no fantasia, no confabulation and no bodily hallucinations. Tests of thought disorder revealed chiefly alolia but some paraphasia. In view of the fact that she was then aged 77 years, these findings are probably of little importance.

Only two cases of Progressive Somatopsychosis (Hypochondriacal Hallucinations) were found. The ages of onset were 33 and 38 years. No family history of mental illness was found in either case, unless one includes the fact that one patient's father was described as a heavy drinker.

H.C. was admitted to St. Nicholas Hospital on 25 March, 1929 when aged 38 and having suffered from mental illness for about five years. There was no family history of nervous illness. On admission she is reported as being restless, noisy and quite irrational in her talk. At this time she imagined she had her two children in bed with her and talked to them. In 1930 she complained of electrical persecution by the nursing and medical staff and accused her doctor of murdering her mother. In 1935 she claimed that her bones were affected by electricity and she was damaged with electricity by a man called Bobby whose name she heard shouted by spiritualism. In 1937 she said "That Chatham Cleaner Man has been in and I am dying". Later she complained that her eyes were tampered with. In 1941 she asserted that people were "busy at night drawing the sinews" in order to annoy her. In 1950 she complained of a machine working on her hand making it heavy. In 1951 she was found to have a cataract and a detached retina in the right eye and to have myopic changes in her left eye. She said that the air was making her go blind, making her head smaller and her body shrink. In 1955 she complained that her arm and shoulder were damaged and said she heard a man talking in the night and said "trying to put it right, I suppose". She admitted hearing voices all the way through her illness but at no time were the voices prominent or was she seen talking to them. Her bodily hallucinations were complained of more consistently and determined the form of her illness. In tests for thought disorder she showed paralogia and tended to see a personal meaning in the questions.

Only one doubtful case of Progressive Autopsychosis was found. Another markedly expansive patient occurred in this series but could not be placed in this subgroup as she had clear-cut hallucinatory voices and bodily hallucinations. This one case of Autopsychosis is open to doubt, as no record of grandiose ideas could be found in the case notes until the 35th year of illness and in the 38th year condylomata were found on the vulva and the Wassermann and Kahn reactions were found to be positive. She was treated with penicillin; no signs of general paralysis were to be found.

This patient, M.A., was admitted to the Northumberland County Mental Hospital in 1917 following the birth of an illegitimate child. The certificate on 27 September, 1917 states "She believes she is responsible for the war. Thinks people are talking about her. Recently confined." No notes are available again until 1921 when there is a note "Cannot tell age. Says she is a married woman but died and was brought to life. Mutters to herself." In 1924 she is reported as childish and excited and quarrelsome at times. In 1930 the notes say "Simple and childish." Year 1925, "Apathetic". In 1934 "Solitary, talks to herself." In 1943 "Childishly stubborn and quick tempered at times. Usually uninterested, rather vacant and apathetic." In 1949 the notes state "Simple and childish. Good laundry worker. 'This used to be Holyrood Palace but has since been christened Gosforth Hospital.'" In 1951 when asked her age she said "I only know it as 21 because I have been through all my eternity and so I cannot remember years." In 1953 she claimed that she had been married 10 years and that she was on the stage as Vesta Tilley (a well-known Edwardian variety actress) when she was young. In 1954 condylomata were discovered and the blood W.R. and Kahn were then found to be positive. She was treated with penicillin. In 1955 when I first saw her she told me she was Queen Alexandrite and that King Edward was her uncle. She asserted that four years before while in the hospital chapel she was called out from the other patients by the Archbishop and crowned as Queen of Scotland. In tests of thought disorder she showed alolia, some paralogia and some paraphasia. It was impossible to determine when this patient's grandiose delusions began. It is possible that the reference to Holyrood Palace in 1944 indicated the presence of a grandiose delusion.

This completes the typical paranoid forms and now we must consider the combined forms. Firstly there are the combinations of paranoid subforms of which eleven examples were found.

Five of these were combinations of Phantasiophrenia and Verbal Hallucinoses. In all these patients there were well-marked fantastic delusional formations but clear hallucinatory voices were also found and could not be considered to play a minor role in the total clinical picture. The age of onset ranged from 29 to 63 years; the average age of onset being 45.2 years. One patient had an uncle who suffered from religious mania but no further details of his illness were available.

A typical example of this illness was W.M.D. who was admitted to Fair Mile Hospital, Berkshire, on 13 May, 1943 when aged 50. It appears from the notes that she had been strange for some time but the exact length of the illness on admission was not certain. On admission she was very talkative and protested about her detention. Just prior to her admission she barricaded herself in her house and asserted that she was being persecuted. When her brother effected an entry she insisted on searching him for hidden poisons and even looked under his fingernails for poison. At this time she was also grandiose and insisted that she was the Queen of England. She was transferred to the Old Manor, Salisbury on 30 May, 1943, but was re-transferred to Fair Mile Hospital on 30 June, 1944. By this time she was haughty in her attitude and high flown in her speech. She said her son's address was G.P.O., London and she had seen him many times in the recent past. Actually her only son had been killed in the R.A.F. two and a half years before. She said that her son had been invalided out of the Navy because of too much parachuting and that he was not only a specialist in all sorts of things medical and otherwise in the Navy but was also a specialist in the police. She continued to express grandiose and persecutory delusions based partly upon auditory hallucinations. She began to insist that she was a "detective inspector specialist". In 1947 she said she had been appointed "Detective Inspector Expert" by the Leeds Assizes, but her house was broken into and she was abducted by Rigdon Brothers of Sheffield. She said she was continuing her investigation into the plot by means of wireless and T.V. On 18 September, 1947 she was transferred to St. Nicholas Hospital. At this time she was voluble and spoke rapidly and forcefully. She said "My son has been thrust into the vortex of the educational ecclesiastic of the specifics of the world" and "God only knows why I am in hospital but my name is W.M.D. a detective expert initiated by my son when he was 19 years old into the Chatham Division of Scotland Yard". There was no essential change in her condition and when seen by me on 15 November, 1955 she was aggressive in manner and declaimed rather than conversed. She insisted I was Admiral Beebak and the content of her remarks was made up of fantastic persecutory and grandiose delusional ideas. She admitted auditory hallucinations which she said were from 129 Office and were the "electrical effects of God Almighty's ether". Her mood was euphoric and she smiled superiorly when her fantastic delusions were denied. In tests of thought disorder she showed paralogia and neologisms. The neologisms appeared to be a paralogic attempt to describe delusional and hallucinatory phenomena. In view of the marked fantasia with misidentifications and the important role played by auditory hallucinations this patient was considered to be suffering from a combination of phantasiophrenia and progressive verbal hallucinosis.

Seven cases of combined Verbal and Hypochondriacal Hallucinoses were found. The age of onset ranged from 21 to 56 years but only two of the patients were under 40 at the time of onset. The average age of onset was 43 years. No family history of mental illness was found in this group of patients. Most of these patients were grandiose to some degree and showed fantastic delusions.

L.L.A. typifies this group. She was admitted to Herrison Hospital, Dorset, in 1945 having been found wandering. She was 51 years old on admission and had probably been ill for less than a year. There was no family history of mental illness. The certificate contains the following statements. "Delusions about Jesus Christ and spirits. Crying and smiling alternately. Told a strange man his wife was ill. Her spirit goes through the bowels of the earth. She hears voices of evil spirits talking to her. Doctors work witchcraft on her and other patients." She was transferred to St. Nicholas Hospital on 26 June, 1946. Since that time she has heard hallucinatory voices which interrupt conversation, to which she talks and occasionally has had screaming outbursts directed to voices. She has also complained of drugs affecting her body and of constantly being sexually interfered with at night. She was seen by me on 21 January, 1956, when auditory and bodily hallucinations were complained of. Later when an attempt was made to administer tests of thought disorder she refused to co-operate.

The only other combination of paranoid subforms was a combination of Progressive (Expansive) Autopsychosis and Verbal Hallucinoses.

This patient, A.C., was admitted to St. Nicholas Hospital under Section 20 of the Lunacy Act on 10 June, 1954 when 48 years old. She had entered the Station Hotel, Newcastle upon

Tyne and insisted that she was Queen Victoria and was master of the hotel. There was no family history of mental illness. The patient had worked as a shop assistant until 14 years before her admission when she developed "nervous debility" and never worked again. Her sister with whom she lived was unable to give any details of the "nervous debility" and it appeared that this patient had been quietly suffering from schizophrenia for fourteen years and then because she acted on a grandiose delusion she had to be admitted to hospital. She settled down immediately after admission. She kept herself neat and tidy. She was euphoric and fatuous and blandly expressed her grandiose delusions without being upset by contradictions. She usually asserted that she was Queen Victoria but her assertions about her relatives were contradictory. Thus she said her mother was Queen Mary and she was waiting at the Station Hotel for the Duke of Edinburgh. On one occasion when asked if she were an important person she said "I am Queen Victoria . . . Mr. George Stephenson . . . Mr. Joseph Cowan". She complained of hearing voices and attributed this to an "electric ear". She said the voices talked to her and told her she was Queen Victoria on occasions, on another occasion she said the voices told her that she was George Stephenson. She began work in the laundry after her admission and was home on leave every weekend. She was discharged in mid-1956 and has attended St. Nicholas Hospital as a day patient and works regularly in the sewing room. Tests of formal thought disorder showed mainly alogia, some paraphasia and confabulation. In view of the continuous auditory hallucinosis which supplied the grandiose colour to her delusions it was felt that this patient could not be classified as a typical Progressive Autopsychosis.

Combinations of paranoid schizophrenias with confused schizophrenias were found. There were three cases of Combined Phantasiophrenia and Incoherent Schizophrenia, one case of combined Phantasiophrenia and Paralogical Schizophrenia, and one case of combined Progressive Autopsychosis and Incoherent Schizophrenia.

The age of onset in the three cases of combined Phantasiophrenia and Incoherent Schizophrenia ranged from 24 to 34 years and the average age of onset was 35.3 years. These patients had gross fantasia associated with incoherent thought disorder and auditory hallucinations. There was a family history of mental illness in one case where the patient's mother had committed suicide.

E.C. is a good example of this combined form. She was admitted to St. George's Hospital, Morpeth, on 15 May, 1933, and transferred to St. Nicholas Hospital on 1 April, 1935. She had hallucinatory voices and persecutory delusions on admission. She settled down following admission but then had short periods of overactivity and noisiness. She was usually euphoric but occasionally became awkward and unco-operative. Her mental condition in 1955 and 1956 when I saw her frequently was the same as it had been for the previous ten years. She was continually auditorily hallucinated. She was usually euphoric and co-operative but occasionally would become suspicious and non-co-operative. She kept herself clean and tidy but usually decorated herself with pieces of paper, cardboard and wool. She laughed, giggled and simpered during interviews. She spoke rapidly in an indistinct Northumbrian accent. The content of her talk was fantastic and grandiose but it was so incoherent and indistinct that at times it could hardly be understood. She would continue talking once started and the flood of incoherent irrelevancy could not be stopped. On one occasion she was told the parable of the Prodigal Son and then asked to tell the parable in her own words. She said "I can just tell you that much that my brother is prodigal this morning. Yes but he comes in William for a meal and when he comes in he was stopped and this is Mrs. Oliver . . . David Oliver. She's David Oliver she is a woman and also makes a . . . (It was impossible to record any more for a few seconds) . . . I am Mr. David Oliver and Mr. Bembridge. Mr. David Oliver I are. Take my photo. I am the Town Clerk—listen—I'm a Lancashire—listen—I'm a Lancashire man. Yeess." It appeared from other interviews that angels talked to her, somebody had been murdered in confinement, something had made her blood brown and there had been a Massacre of the Innocents. She refused to co-operate in tests for thought disorder.

The patient with combined Phantasiophrenia and Paralogical Schizophrenia showed fantasia, paralogical thought disorder and persecutory beliefs.

This patient, A.T.H., was admitted to St. Nicholas Hospital on 15 August, 1940. She was 35 years old on admission but there was a history of attacks of confused speech for the previous six years and during these episodes she would get out of bed at night in order to clear her throat. Her father is recorded as being a heavy drinker but there was no family history of severe mental disorder. The admission note states "Chatters to herself, shouts at times and becomes unsettled. She talks of a wire on her head stopping her breathing, that God made her a 'Head girl of sense' and that people on Earth are Gods." In 1941 she was reported as doing ward work but chattering to herself and rambling in conversation. This state of affairs continued, in addition she was reported as being noisy and shouting at times. In 1947 the notes

state "Says she has been here 140 years and she has no proper age. She is frequently abusive and noisy and apt to be impulsive." In 1948 "Believes she came here because of a cold 250 years ago". In 1950 notes state "Has been here 5,000 years" and go on to say that she is a good laundry worker. In 1952 she complained of lice in her hair and dickies (colloquial word for lice) in the air around her. She claimed to be able to see and hear these dickies. In 1956 when asked why she did not go to the hospital entertainments she said it was "because the dickies were running around". She equated the dickies with neurasthenia or influenza which flew about and gave people colds. She said "they must affect your throat and chest. I keep myself clean by sweets, orange juice and sugar and coffee." When I interviewed her on 24 November, 1955, she talked of "a gas thing seeking blood and coppies out of you". She explained that coppies were dickies. She talked of living thousands of years, being trapped in a machine and being raised from the dead. In tests of thought disorder she showed paraphasia and paralogia.

The patient with combined Autopsychosis and Incoherent Schizophrenia had marked grandiose ideas, auditory hallucinations, incoherent thought disorder and destructive behaviour. This patient had a maternal aunt who had died in a Mental Hospital but further details of this relative's illness were not available.

This patient, H.K., was originally admitted to Graylingwell Hospital on 14 May, 1929. She was then aged 39 years and there was a history of periods of odd behaviour at about four-monthly intervals for the previous four years. The notes immediately after admission state "She is noisy and restless constantly chattering and waving her arms. She has auditory hallucinations, talking to and swearing at unseen voices." She apparently improved and was discharged on 2 August, 1929. She was admitted to Netherne Hospital on 6 June, 1933 on certificate which stated "She has hallucinations of hearing. She imagines she hears voices and frequently screams out in response to them. Her conversation is obscene. She is truculent and resistant towards the nurses in attendance." She was transferred to Graylingwell Hospital on 3 October, 1933 and later to several other mental hospitals, finally being transferred to St. Nicholas Hospital on 5 July, 1949. At this time the notes state "Muttered whispering with no relevance to the interview. Says she is related to Royalty through her sister and her father was King William." Her behaviour has been unchanged since that date. She has usually been euphoric and has sat muttering to herself. She has also been very untidy and destructive. She frequently tore her clothes and would light and destroy several cigarettes before finally lighting one and smoking it. She chattered frequently to her voices and often looked at the ceiling and smiled while talking in that direction. She would not admit to hearing voices and would never give the content of the voices. She was grandiose in manner at times and would posture. She insisted that she was Queen Wilhelmina of England, that her father was King William of England and Mr. Richardson and that King Edward VII was her stepfather's father. She also claimed that her sister who died at the age of three months was the wife of the Prince of Wales. She was very incoherent and her grandiose delusions were selected from material much of which was incoherent and incomprehensible. Her replies to questions were often incomprehensible or beside the point. Thus during one interview she stood up suddenly and when asked why replied "Because I am a mistress". She would not co-operate in tests of thought disorder. In view of the auditory hallucinations, the incoherence and the marked grandiose delusion she was considered to have a combined Autopsychosis and Incoherent Schizophrenia.

Seven true atypical paranoid schizophrenics were found. The age of onset ranged from 21 to 44. Four patients had a family history of mental illness requiring mental hospital admission. Kleist called this group the Progressive Reference Psychosis and subdivided it into three groups, Self Reference Psychosis, Signification Psychosis and Circumscribed Delusional Psychosis (Kleist, 1949).

A.H.A. is a good example of Self Reference Psychosis. She was admitted to St. Nicholas Hospital as a voluntary patient on 16 July, 1948, when aged 37 years. About the age of 32 her husband died and shortly after this she began to believe that the curate at the Parish Church was fond of her. She took part in amateur dramatics connected with the church and believed that the other girls at the church were jealous of her. She gradually became dominated by ideas of self reference and persecutory ideas. As her mother believed many of the patient's delusions it is difficult to get a clear picture of the development of the psychosis: About five months before her admission she accused her fellow employees at the Ministry of National Insurance of putting bromide in her tea. On admission she had an elaborate system of delusional and hallucinatory experiences which she said had been present since her grandmother died eight years before. She claimed that her mother had had similar experiences for 50 years. This was not confirmed although her mother believed firmly in the patient's

persecutory delusions. The patient said she heard Jimmy Knox and Sir Claude Gibb calling her an abortionist. She believed her food was drugged or poisoned and was very hypochondriacal. In the next few months she improved but according to the notes she was deluded. On 20 November, 1948 she was discharged relieved. She was re-admitted on 14 April, 1950. She gave several different names on this occasion, claimed she was the Virgin Mary, that she had two husbands living and that she was entitled to the name of Du Maurier. From this time on she mistook identities, changed her own identity, complained of persecution, had auditory hallucinations and was frequently very hypochondriacal. Her thoughts became very disordered and she tended to declaim persecutory and grandiose nonsense with many neologisms, and paraphasia and paralogia. She usually smiled when she saw me and at the beginning of an interview, however, once she was spoken to she fiercely declaimed her incoherent nonsense in a rather frightening aggressive way, but in fact was never violent to the staff or patients. She co-operated in tests of thought disorder and showed paraphasia, paralogia and neologisms which appeared to be technical expressions of delusional and hallucinatory experiences.

E.B. is the only example of Circumscribed Delusional Psychosis found in this series.

She was admitted to the Crichton Royal on 24 May, 1941, when aged 43. Her uncle had been in a Mental Hospital but his notes could not be traced. She had ideas of guilt, thought she would be punished and was restless and agitated following an air raid one month before. She improved but one week later following another air raid she became depressed, thought that there were transmitters under the bed and people were listening in for her to give away information. On admission she was agitated with marked ideas of self reference. She felt everybody knew about her and the police were coming to take her away. She improved with E.C.T. but relapsed quickly. When left to her own devices she tended to be depressed, inactive, somewhat retarded and depressed. She was discharged on 17 August, 1942, and lived at home for nearly five years until her admission to St. Nicholas Hospital on 30 May, 1946. She behaved strangely at times but finally became filthy and neglected herself. On admission she was deluded about being married. She insisted she was about to marry. Later she insisted that she was married to John Robert Gillespie and that she could hear him talking to her. She insisted she was being kept from her husband. These delusional and hallucinatory experiences have persisted and she has become very aggressive and violent at times. From 1949 to 1956 she received over 200 treatments with E.C.T. for her violent episodes. Later she was given reserpine and became quiet and well behaved. In 1954 she showed grandiose delusions which have continued.

It will be noted that there were 21 cases classified as typical forms and 18 cases as combined forms. In many cases of typical paranoid schizophrenia mild features of the other paranoid forms were found. The distinguishing features of the first five paranoid forms are fantasia, confabulosis, hallucinatory voices, bodily hallucinations and delusions of grandeur. Often one or more of these features were found to a minor degree in a typical paranoid form. It did not appear to be legitimate to consider the individual subforms as being sharply delimited from one another. The combined forms could best be regarded as transitions between the typical forms.

One could postulate a pathoplastic factor determining the paranoid features of the illness and other factors determining the form of the paranoid illness. The average age of onset in Paranoid Schizophrenia is higher than other groups, thus in Kleist's series it was 38.7 years (Kleist, 1949) and in the present series 32.1 years. One could postulate that when the schizophrenic process occurs in a mature brain, the inherent stability of the nervous system causes a delusional illness dominated by delusions and hallucinations with fair preservation of the personality. The precise role played by hallucinations and delusions could then depend upon the type of imagery and fantasy which the patient normally has. Thus if the patient has marked auditory imagery then auditory hallucinations will dominate the clinical picture. Thus two of the patients with Verbal Hallucinosis were deaf and one was a Greek from Asia Minor whose knowledge of English was poor. This patient spoke very poor English and when asked about her knowledge of the language she said with a smile that it was all right because everyone in the hospital spoke "pure Greek".

All three of these patients had a desire to hear the spoken word and the Schizophrenic illness allowed them to do so. If objective tests for the dominance of different sorts of imagery could be devised then one might be able to isolate the pathoplastic factors in paranoid schizophrenia.

The Progressive Reference Psychosis might be accounted for on the basis of the severity of the schizophrenic process or the presence of some other pathoplastic factor.

THE CATATONIAS

Eighteen cases were classified as suffering from catatonia and Table III gives the distribution of these cases among the different subgroups. It will be

TABLE III
*Catatonia*s

(a) Typical Forms:

								Cases
Speech-inactive catatonia	..	..	..	..	..	..	..	4
Speech-prompt catatonia	..	..	..	..	..	..	..	1
Parakinetic catatonia	..	..	..	..	..	..	..	1
Prosectic catatonia	..	..	..	..	..	..	..	2
Stereotyped catatonia	..	..	..	..	..	..	..	5

(b) Combined Forms:

Prosectic catatonia and incoherent schizophrenia	..	..	..	..	..	..	..	1
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(c) Atypical Extensive Forms:

Iterative catatonia	..	..	..	..	..	..	..	2
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(d) Other Forms:

Unclassifiable	..	..	..	..	..	..	..	2
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noted that no cases of akinetic or negativistic catatonia were found, however, one case of negativistic catatonia in St. Nicholas Hospital was known to the author, but she belonged to that part of the original series which could not be investigated fully owing to lack of time.

Four cases were classified as suffering from speech-inactive catatonia. The age of onset in these cases ranged from 17-25 years with an average age of 19-2 years. No family history of mental illness was found in these cases.

The patient, D.M., is a good example of this illness. She was admitted to St. Nicholas Hospital as a voluntary patient on 30 October, 1944 when she was 26 years old. She had had an alleged heart attack in January, 1943 since when she had tired easily and had difficulty in getting out of bed in the morning. There was no family history of mental illness. On admission she was described as dull and depressed and smiling childishly at times for no reason. She said that everything she ate was sticking in her stomach and could not pass through. She believed that her stomach filled up with water. Subsequently she appeared to be dull and unresponsive and on 9 November, 1944 was found to have urinary retention with overflow. She was given E.C.T. and improved. She was discharged on 2 August, 1945. She was seen at intervals as an outpatient and gradually became more and more inert. Finally she sat about motionless and refused food. She was re-admitted on 5 April, 1948, and responded to E.C.T.; in that she was quite well behaved and co-operative although still obviously ill. She had a course of 30 Insulin Comas. However, on 1 December, 1948, the notes state "Solitary, has to be dressed. Sits with head bowed at meal times toying with her food." Since this time she has usually sat mutely with the head bowed. She would look at anyone who spoke to her and half smile but rarely replied. At times she would suddenly strike another patient or throw a plate on the floor and then lapse into immobility. She would carry out simple commands slowly at times but at other times the movement would be arrested half way or tentative movements could be seen. Passive movements would easily be carried out. She offered no resistance to being pushed into activity. She would allow herself to be taken to the toilet, put at table, and so on. Limbs tended to maintain the positions they were placed in by the examiner for a few seconds and then would slowly sink to a position of rest. On one occasion an arm was placed in an unusual position and did not fully return to rest within five minutes. These motor symptoms could always be

abolished by 2 or 3 treatments with E.C.T. but would return within ten to fourteen days following E.C.T. In view of response to E.C.T. a classical Freeman and Watt prefrontal leucotomy was carried out on 9 September, 1955 by Mr. L. Lassmann. There was an immediate improvement with loss of motor symptoms, but within three weeks her condition was the same as before the operation. When last seen by me on 6 September, 1956, her condition was substantially unchanged.

One case could be classified as speech-prompt catatonia.

This was the patient E.M. who was admitted to St. Nicholas Hospital on 8 July, 1929, when 30 years old. There was no family history of mental illness. The duration of the illness is not recorded but as lactation was given as a cause of illness it can be assumed that she had not been ill longer than a year or so. The notes on the mental state on admission say "She talks about her name being put about, that she is pointed at, that people frown at her, that they talk about her. She can tell this from a certain way they look at her." For some years after admission the notes say that she was childish, rambled in speech and was irrelevant. In 1937 she is reported as working in the dining hall. In 1940 there is an entry "Rambles in talk and makes completely irrelevant replies". In 1942 there is a record of a period during which she was restless and unsettled and was striking patients and staff impulsively. In 1948 the notes say that she would do ward work if coaxed. When seen by me on 23 April, 1956, she sat quietly and answered all questions promptly. Her face was devoid of expression except for an occasional slight foolish smile. She turned towards me and never tired of answering questions; her replies were usually irrelevant. Thus when asked her name she gave me irrelevant replies and the following conversation occurred: "(Q. Where does the name May (her assumed name) come from then?) A. From the heated weather. (Q. What do you mean by that?) A. One night it was warm. It was warm. I did not know what to do . . . So I said May had less letters in. (Q. How has hot weather got anything to do with the number of letters in your name?) A. That was the way it was soothed." She showed no evidence of perseveration of posture. She was solitary, never mixed with other patients, never spoke spontaneously and was completely unsociable. She worked sloppily on ward work and in the laundry but showed no initiative. She showed no evidence of auditory hallucinations.

Only one case of parakinetic (buffoonlike) catatonia was found.

This patient, A.W.E., was admitted to St. Nicholas Hospital on 25 September, 1933, when she was aged 22 years. There was no family history of mental illness. There was a history of illness for four years before admission. She had become silly in behaviour and very slow in activity. She talked to unseen people and began to believe she was going to have a baby. On admission she wept when spoken to and said she would die a horrible death and that her blood stream had stopped, making her terribly ill. She had scratched her legs and breasts with a broken button. She said she was sure she was going to die and if she did not die she would be turned into an animal. In 1933 and 1934 she had frequent attacks of agitated depression. On 25 August, 1934, she expressed a grandiose delusion that she was an heiress called Lady E. In 1935 she was described as "Childish and affected, has exalted delusions, uses neologisms and unintelligible jargon, is erotic and lacking in self control and is a runaway". From this time until 1940 the notes mention that she is facetious, giggles, is affected and has shy phases. In 1943 the notes state "Talk affected, rambling, deluded and very irrational, e.g. 'I said they were almond sweets'." In 1949 there is a note "Has continual choreiform movements of hands". In 1950 the notes say "She mutters to herself continually, adopting queer strained postures". In 1952 the notes state "Very apprehensive. Talks to herself in a jumbled stream of irrelevant phrases and will not answer the simplest questions."

When seen by me on 22 September, 1956, she answered questions incoherently in a jerky manner. Some word or words relevant to the reply could be extricated from the incoherent material. She sat in a crumpled position with her back slightly bent and twisted with her left shoulder lower than her right. She walked in a crab-like way. She got up, walked about, touched the door handle and sat down again several times during the interview. She showed short odd jerky movements of her body. Her behaviour in the ward was frequently disturbed. She tended to run up and down the ward usually sideways and would half turn to a call but run on. At times she would knock all the dishes on to the floor. She stole food from other patients at meal times. At times she would handle all objects within reach. If she could get to a water tap she would splash the water about. She usually was overactive for four to seven days and then would be quiet for a few days. In her overactive periods senseless random sudden short-lived assaults were made on patients and staff.

Two patients were classified as suffering from prosectic (proskinetik) catatonia. The ages of onset were 35 and 27 years. One of these patients had a paternal grandmother who had a nervous illness in her sixties but recovered

and lived to the age of 90. No clinical details of this illness were available. No family history of mental illness was found in the other cases.

A.E.M. is an example of this illness. She was originally admitted to the Crichton Royal, Dumfries, on 18 June, 1927, when 35 years old with an illness which had begun six months before. There was a family history of mental illness in that her grandmother had a nervous illness in her sixties but allegedly recovered completely. On admission to Crichton Royal the patient had auditory hallucinations, ideas of reference, thought disorder and appeared depressed. There was no change in her condition until 1931 when it was stated that she was becoming "enfeebled mentally". In 1935 the notes state that she was "demented, idle, apathetic, intermittently noisy". In 1937 the notes state "periodically hallucinated, out of touch with her surroundings, remains quite unoccupied, showing gross thought disorder, being quite unintelligible". There was no change in her condition reported and in January, 1947 she was transferred to St. Nicholas Hospital, Gosforth, when the transfer notes at the Crichton Royal state "fatuously pleasant at interview, whispering irrelevant nonsense, demented". On admission to St. Nicholas Hospital the admission note reads "She understands things which are said to her and responds but cannot carry out connected conversation. Much of what she says is quite unintelligible and at times she becomes very noisy shouting at some imaginary person." From that time on there are records of her muttering to herself at times, shouting to voices occasionally, hooting at night and hiding herself at bedtime. When seen by me on 14 February, 1956 she would grunt and mutter when spoken to. If she stopped speaking she could be started again by a nod or a grunt or by irrelevant words in French, German, Italian or Russian. She never tired of replying. She had short-lived periods of excitement in which she handled and touched all accessible objects.

Five patients were classified as suffering from stereotyped (Manneristic) catatonia. The age of onset ranged from 21 to 30 years with an average of 26.4 years. Two of these patients had a family history of mental illness. Each had a maternal uncle in a mental hospital and one had a mental defective sister.

E.S. is a good example of this illness. In 1943 she became ill when aged 21 and serving in the W.A.A.F. Her original complaint was fear of travelling. She was admitted to St. Andrew's Hospital, Northampton, and while there surprised her parents by writing to them and denying that her surname was S. On 27 July, 1944, she was admitted to St. Nicholas Hospital, Gosforth. She was then showing grandiose delusions. She claimed to be the Queen of England and to be engaged to H.R.H. Prince Baltimore and also to be the Duchess of York. She behaved in a childish irresponsible way and on one occasion attempted to run away by climbing a wall. She improved and was discharged on 19 April, 1945. She was re-admitted to St. Nicholas Hospital on 4 October, 1948, when she had doubts about her relationship to her parents and believed she had been hypnotized by men. She was treated with E.C.T. but became very confused. In 1949 she was treated with insulin coma therapy and had 30 comas with some improvement. However, she began to express delusional ideas again, saying that she was in hospital on a bailiff's order and that the prostitute industry was interfering with her in some way. In 1951 the notes state "becomes oblique and irrelevant in her conversation." She was deemed to have lost her volition and was therefore certified. This year there is a note that she is working in the laundry. I interviewed her on 24 November, 1955. She sat sideways on the edge of her chair. She bent forwards and sat up again continuously. She rubbed her hands together after placing them between her legs to do that. She also clapped her hands, nodded her head and swayed her body. She fluttered her eyelids continuously, smiled and raised her eyebrows. She usually looked at the floor but looked up at me at times and then away. She bit her lower lip and at times rubbed her eye. She muttered continuously whether spoken to or not. Her replies to questions were mainly incoherent except for those simple questions such as "What is your name?" In the ward the patient tended to walk round in circles clutching her skirt. She was doubly incontinent and often played with her faeces and with nasal mucus. She appeared to be obviously auditorily hallucinated and spoke to voices. She did not have noisy outbursts.

Two cases of iterative catatonia were found. The age of onset was 20 years in one case and under 32 years in the other. The exact age of onset in this last case was uncertain. In one case there was a family history of mental illness. The patient's father died in a mental observation ward and a paternal aunt had been in a mental hospital. No details of these illnesses were available.

This patient, J.C., illustrates this subgroup. She was admitted to St. Nicholas Hospital on 1 May, 1930, when aged 32. Her mother said that she had always been quiet and dull and often walked in a peculiar way and when asked why she said she was measuring her feet. At night she would walk about her bedroom instead of going to bed. On admission she was listless,

lethargic, apathetic and lacking in initiative. She was depressed and lugubrious and wept without cause. In June, 1930 she was reported to have improved and was working in the laundry. From this time on she alternated between states when she was dull and apathetic, but would work under supervision, and excited states when she was overactive, talkative. She showed thought disorder on one occasion when she said "My feet never hurt in this place, but this place has hurt my feet". In 1950 there is a note "attacks of agitated depression". In 1953 the notes state "Dull, rigid and retarded. Sometimes co-operative, sometimes negativistic." In 1954 there is an entry in the case notes "Subject to noisy outbursts, impulsively breaks windows". During 1955 I saw this patient in excited and quiet states. On 24 April, 1956, I interviewed her during an excited state. She perseverated and repeatedly said "They say it is Jane Ann Armstrong who is doing wrong things", "They say I have kept all my things", and "Nurse Swindel says I must keep things". She held a handkerchief and two hair slides in her right hand. When allowed to leave the office she walked up and down the day room repeating the stereotyped phrases mentioned above. She would reply to simple questions such as stating my name, but would perseverate the answer until she fell back on to one of the other phrases mentioned above. She was reported to make repetitive movements by the nursing staff, but I did not see any such movements.

Only one combined form was found and this was a combination of a prosectic catatonia and incoherent schizophrenia.

This patient, L.B., was admitted to the Northumberland County Mental Hospital on Certificate in 1919, aged 19, with a history that she had been restless and sleepless for a year when she was 17 years old and was in the mental ward of Oldham Workhouse for one year. The certificate in 1919 stated that she was "strange in manner, impulsive and violent. Passionate and vicious." In 1921 the notes state "Speech unintelligible and rambling. Noisy irritable and impulsive." "Dull and stupid and resistive frequently tries to take off her clothes. Chatters and mutters to herself. Is wet and dirty." In the next ten years she alternated between a state when she was dull and stupid and did not talk to others and a state of excitement. In the 1930's the notes frequently say that she will not talk, but sits grinning to herself. At times she is described as wet and dirty. In 1942 she is recorded as doing some ward work. In 1943 the notes state "excited and resistive when her rubbish hoards are investigated". In 1946 she is reported to be deaf and to have corneal nebulae interfering with her sight. After this there are notes of impulsive outbursts, muttering to herself and snatching things from others: when seen on 24 January, 1956, she sat with her head bowed slightly and her back slightly bent. She refused to carry out orders such as "Touch your nose" but did shake hands when I offered my hand. She looked at me most of the time and only occasionally looked away. She rocked herself to and fro and rubbed the table with a dirty rag. She replied to questions in a thick guttural voice and most of her speech was incoherent. She finally stood up and muttered and one of the words she said indistinctly sounded like "Finished". She then wandered round the room. The ward sister reported that she was usually inactive but would suddenly attack other patients regardless of their ability to fight back. At other times she was noisy and destructive. When excited she would handle and touch all available objects in the ward. She was frequently seen to talk to hallucinatory voices and often hallucinated during conversation. It appeared that the voice of God told her to attack others and steal. It was considered that she did turn towards the examiner but her deafness and poor vision masked the prosectic speech features to some extent. Her handling and touching of objects would be in favour of prosectic (proskinetik) catatonia. Her incoherence and frequent auditory hallucinations which dominated the clinical picture in the less excited periods appeared to be atypical of prosectic catatonia and it was thought that an incoherent schizophrenia could best account for this.

Two patients could not be classified. One of these had had two prefrontal leucotomies and a very large amount of E.C.T. It was considered that her original condition was probably speech inactive catatonia and the clinical features had been obscured by brain damage. The other patient suffered from a catatonic illness with extremely violent behaviour and self-destructive behaviour. She had had a prefrontal leucotomy and was receiving E.C.T.

The classification of the Catatonic patients was not easy as many of these patients had had long periods of "maintenance" E.C.T. which masked symptoms and altered the usual variations of the illness such as outbursts of excitement.

The average age of onset was 24.4 years, but the range was from 17 to 35 years. In seven cases the age of onset was below 21 years, in six it was between 24 and 27 years and in five it was over 30. However, in two cases the age of onset was taken as 30 because the age of onset was uncertain, the age of admission being taken as the age of onset.

In the four speech inactive patients the age of onset ranged from 17 to 25 years, and in three of these cases it was at 17 or 18 years. The age of onset of the five stereotyped (manneristic) catatonics was from 21 to 30, and in four cases it was 25 years or over. Despite the wide scatter the age of onset in Catatonia is on the whole considerably below that of paranoid schizophrenia. One could argue that the schizophrenic process in an immature brain may produce catatonia if some other pathoplastic factors are present. If one assumes that this second factor is a psychomotor expressive mechanism, then the age differences between speech-inactive and stereotyped catatonia could be used to explain the differences between these forms. It could be argued that the mutism and co-operation of the speech-inactive is more primitive than the stereotypy of the stereotyped catatonia.

THE CONFUSED SCHIZOPHRENIAS

Apart from the combined forms of confused schizophrenia with paranoid schizophrenia or catatonia twenty-one cases of confused schizophrenia were found. The distribution of these cases among the different subforms is shown in Table IV.

TABLE IV
Confused Schizophrenias

	Cases
(a) Typical Forms:	
Schizophasia	4
Incoherent schizophrenia	10
Paralogical schizophrenia	2
(b) Combined Forms*:	
Incoherent schizophrenia and phantasiophrenia	3
Incoherent schizophrenia and autopsychosis	1
Incoherent schizophrenia and prosectic catatonia	1
Paralogical schizophrenia and phantasiophrenia	1
(c) Atypical Extensive Forms:	
Atypical confused schizophrenia	5

* These cases have already been included in Tables II and III.

Four cases of schizophasia were found. These patients all showed gross thought and speech disorder but were good workers and made good relationships with the nursing staff. The age of onset of illness ranged from 31 to 36 years, the average age of onset being 33·75 years. A family history of mental illness was found in two of the cases. In both cases a brother had committed suicide and in one case the brother was described by the relatives as suffering from religious mania.

F.A. is a good example of schizophasia. She was admitted to St. Nicholas Hospital as a voluntary patient on 12 February, 1955, when aged 41 years. She had been a voluntary patient in Winterton Hospital, Sedgfield, in 1945 and on 6 April, 1946 she was certified. On that occasion the note on admission states "She is dull and retarded and shows much dissociation and disorientation. She is confused and unable to give a connected account of herself. She is auditorily hallucinated and talks to herself. She has no proper appreciation of her condition." She was quiet and well behaved and after six months she was discharged from hospital. Her husband was frightened of her as he said she had been very violent. She therefore went to live in her parent's home in the same small mining village where her marital home was. Her family was ashamed of her mental illness as she was the only member of the family who had ever been in a mental hospital; although her brother committed suicide for no obvious reason at the age of 19. She was sent to London to relatives and for the next nine years she held innumerable jobs in London, usually each job lasted from a few weeks to a few months. At somewhere about six monthly intervals she would return to her family penniless and after a

week or two she was given her train fare to London by her mother and sisters and went back to her previous existence in London. In late 1954 she returned to her mother and was told to go and then lived with her eldest son who did not really have enough room. I saw her as an out-patient in mid-January, 1955 when she had what at that time appeared to me to be gross formal thought disorder and was unable to give a coherent account of herself except to say that she was tired. On 12 February, 1955, she arrived at St. Nicholas Hospital having been turned out by her son and having spent all her money on her fare to the hospital. I had tried to avoid admitting her because I felt she would never leave the safe haven of hospital once she got in. However, there was no alternative but to admit her as a voluntary patient. Since her admission until last seen in December, 1956 this patient has shown gross difficulty in verbal expression, has worked well for periods of a few months and had short-lived agitated depressed spells when she has wished to leave hospital but has realized that this was impossible because her mother and sister would not have her home. Outside these spells she was well behaved and understood and carried out orders very well. She made relationships with the nurses and patients. I got to know her very well and with patience it was always possible to get to understand the main ideas which she wished to express. Sometimes this was not easy. Thus she talked about signing a form and the following conversation occurred: "(Q. I don't understand what you are talking about. What is all this about signing a form?). A. Well the purpose was my colour really. (Q. The purpose was your colour?) A. Yes. (Q. That does not mean anything to me.) A. Well it does to me, I can say. (Q. The purpose was your colour? I don't follow that.) A. Well whatever I did I had my colour in mind and—er—I mean what ever I did I had a purpose with it. (Q. What do you mean by your colour?) A. Well . . . my periods." What she was trying to talk about was an application to the National Assistance Board for assistance, and as she was a married woman some claim might have to be made on her husband and at least she had to give details of her marriage. She used the word colour to mean her periods in order to express the idea that her marital affairs were under discussion. In formal tests of thought disorder she showed paraphasia and paralogia.

Ten cases were classified as incoherent schizophrenias. In nine of these patients the age of onset was between 30 and 40 years and in one patient it was 51 years. The average age of onset was 36·3 years.

Five of these patients had a family history of mental illness. In one case there was an entry in the case notes that an uncle was insane, but no further details could be obtained. Another patient's mother died in a mental hospital as did a number of other relatives but no further details could be obtained from the patient's sister who was the sole informant and herself a very eccentric woman. Another patient had a brother in a mental hospital but no further details could be obtained. A fourth patient had a mother who was in St. Nicholas Hospital for many years. She was a hardworking trustworthy patient who from time to time had mild agitated depressive illnesses which responded to E.C.T. and she showed no evidence of schizophrenia. A fifth patient came from a family heavily loaded with schizophrenia. Her niece was an in-patient in St. Nicholas Hospital, her mother and an uncle suffered from schizophrenia and died in St. Nicholas Hospital, her father died in St. Nicholas Hospital while suffering from a depressive illness probably due to cerebral arteriosclerosis.

J.M. was admitted to St. Nicholas Hospital on 30 December, 1939, when aged 30 years. She had possibly been ill for two years. She was six months pregnant on admission. She was then depressed and wept at times. She complained that she felt confused and said she was unable to concentrate and everything was wrong inside her head. Her mother had been an inpatient in St. Nicholas Hospital for many years with recurrent depression. On 9 April, 1940 she had a male child in Newcastle General Hospital. On 30 August, 1940 she is reported as "Stubborn, dejected, exalted, oblique and peculiar. Attitude childish." In 1942 she is reported as "often strange in talk" and in 1943 "makes voluble deluded statements and accusations in an excited manner". In 1946 there is an entry "Peculiar in manner, and phraseology, much of her talking and writing is irrelevant". In 1948 she was working well in the laundry. She appears to have had excited periods throughout her illness when she was overactive and sometimes shouted at hallucinatory voices. In quiet periods she muttered to the voices, sometimes gestured and grimaced in one direction as if speaking to voices and sometimes used obscene language to the voices. She insisted that she was married to a senior medical officer in 1947 and since then has repeated this delusion. She also insists that another doctor seduced her in the laundry. Her manner is usually haughty and has expressed vague grandiose delusions such as "the Royalties—the King and Queen keep my people in money". In recent years she has refused to work. Her mother who is a parole patient visits her regularly but the patient

shows little affection and is only interested in the cigarettes she is given by her mother. She tends to refuse to answer questions and give evading answers so that on formal tests of thought disorder she shows what could be mistaken for alogia. However, when she speaks spontaneously she shows gross incoherence and some paralogia.

Two cases of paralogical schizophrenia were found but both were atypical to some degree. The ages of onset were 26 and 29 years. One patient had a mother who died in a mental hospital.

The most atypical patient had short-lived attacks of screaming and impulsive violence which did not appear to have an hallucinatory basis. She had a marked paralogical thought disorder. At one stage of her illness she had a small toy motor car about four inches long and said that as she was about to go to college she needed a car.

The other patient had marked paralogical thought disorder but there was no history of absurd actions based on the thought disorder which is so characteristic of paralogical schizophrenia.

This patient, M.C., was admitted to St. Nicholas Hospital on 3 July, 1933, when aged 35. Her mother died in the hospital and had been an in-patient from 1902 to 1927. This illness appeared to be schizophrenic. According to the history on admission the patient had lost all heart after the death of her husband six years before and had become very depressed, worried, agitated and emotional. She was continually asking the same question about herself and her bodily state. On admission she heard hallucinatory voices of men and women which told her she had done wrong. She also said she heard screams and heard terrible things being said. An attempt was made to employ her in the laundry but she wandered about aimlessly. For the next ten years she is described as dejected, retarded and always mildly depressed and would work occasionally. In 1947 she is described as "dull, rather stupid with delusional ideas and is tense, nervous and apprehensive". In 1948 when interviewed she said "I don't know anything. I am tired. I am an English wage worker." The subsequent notes record that she is incoherent and incoherent in speech. In 1951 she was asked "Do you go to the dances?" and she replied "I am getting my feet attended to". In 1952 she told an interviewer "English abdomens are soft. My brother calls for me but not on visiting days." In 1954 she said during an interview "We've both been vaccinated. Do you ever hear tell of the boys go in when they're coming like. My brother guides me through the law of the Church." She showed paralogical thought disorder in spontaneous speech and during formal testing. There was no recent history of auditory hallucinations. The patient had worked reasonably well in the laundry since 1951 and had been on parole for five years.

Five cases of Atypical Confused Schizophrenia with a shift-like course were found. Four of these patients had a family history of mental illness. Two patients had uncles who had committed suicide. A third patient had a father with schizophrenia and a brother who had been an inpatient in a mental hospital. A fourth patient had a mother who had had a schizophrenic illness, a brother in St. Nicholas Hospital with a schizophrenic illness and a brother who committed suicide. The age of onset ranged from 14-28 years with an average of 23.8 years.

S.E. is a good example of confused schizophrenia with a shift-like course. She was admitted to St. Nicholas Hospital on 8 July, 1929, when aged 33 years. She had a paternal uncle who committed suicide but there is no other family history of mental illness. She had been ill for about a year when admitted. The notes on admission describe her as "impulsive, unreliable and inclined to be violent. Worried, pre-occupied and anxious. Says her memory is bad due to husband's ill treatment." Later in 1929 she was described as suspicious, resentful, abusive and impulsive. In 1930 and 1931 she complained that her food was poisoned. From 1933 to 1936 she is described as "frequently noisy, impulsive, violent and rambling". From 1947 the excited outbursts are reported in which she was irresponsible, hilarious, childish, impulsive and excitedly restless. In recent years she has alternated between excited and quiet periods, each of which has usually lasted three months. During the excited periods she attacks other patients, handles and interferes with all available objects. She is continuously auditorily hallucinated and talks to her voices during conversation. During her quiet periods she shows paralogia and paraphasia. She would appear to be a good example of the affective variety of atypical shift-like confused schizophrenia.

It will be observed that the average age of onset and age range of onset of these subforms and the atypical extensive form are quite different.

In this series the average age of onset of schizophasia (33·75 years) was lower than in incoherent schizophrenia (36·3 years) whereas in Kleist's series the age of onset of schizophasia (35·3 years) was higher than that of typical incoherent schizophrenia (31·4 years) (Kleist and Schwab, 1950).

One could explain the differences in schizophasia, paralogical schizophrenia and incoherent schizophrenia on the basis of differences of intensity of the physiological basis of auditory hallucinosis. In incoherent schizophrenia one could postulate a vivid auditory hallucinosis which inter-penetrated all the thinking of the patient thus producing incoherence. In schizophasia one could postulate that the hallucinatory activity is less and that instead of clear-cut auditory images dominating the patient's mind auditory images become autonomous but are not experienced as sensations. This wealth of autonomous auditory (verbal) imagery would make it difficult for the patient to choose the correct expressions but would not interfere with non-verbal thinking because the individual is not normally dependent on verbal imagery in thinking. The same loosening of auditory imagery in an individual dependent upon verbal imagery for much of his thinking could produce paralogical schizophrenia.

It seems doubtful whether the shift-like atypical confused schizophrenia has much affinity with the typical and combined forms of confused schizophrenia.

THE HEBEPHRENIAS

Twenty-two cases were classified as hebephrenias, that is, the affective change was the most important feature of the illness. The distribution of these cases among the various subgroups is shown in Table V.

TABLE V
The Hebephrenias

									Cases
(a) Silly hebephrenia	..	..	..	..	..	..	..	..	6
(b) Depressive hebephrenia	..	..	..	..	..	..	..	..	4
(c) Apathetic hebephrenia	..	..	..	..	..	..	..	..	4
(d) Autistic hebephrenia	..	..	..	..	..	..	..	..	8

Six patients were classified as suffering from Silly Hebephrenia. The age of onset ranged from 19 to 29 with an average age of 23·3 years. Only one of these patients had a family history of mental illness; her sister was in this series and classified as an apathetic hebephrenic. The patient herself had some atypical symptoms. She had a silly smile and behaved foolishly, but reacted to any attempt to help her such as changing her shoes or clothes or to being brought to an interview by screaming and senseless overactivity like a frightened child.

The patient E.B. illustrates this group fairly well. She was admitted to St. Nicholas Hospital on 6 May, 1954, when she was 37 years old. According to her mother the patient's paternal aunt and uncle were "queer" and had been in "homes" but not in Mental Homes. The onset of the illness was uncertain but since her middle twenties she had been a wanderer. She would take as much money as she could find in the house and all the clothes she could carry and sell them. After some months she would return dirty, dishevelled and penniless. When her mother saw her looking round for clothing, she knew that the patient was about to run away and then she would threaten the patient with a stick and lock the door of the house. She was often on the roads and appears to have been a prostitute of a very inferior kind. When at home she would stand on one leg in the middle of the room while everyone walked round her. Finally she was found on the sands at South Shields by the police and taken to the Mental Observation Ward at South Shields General Hospital. She was unable to give a

clear account of herself. On her admission to St. Nicholas Hospital she was unable to explain what she was doing at South Shields and during the initial interview in the space of a few minutes she told the doctor that she had gone there for a day's holiday, a week's holiday and a fortnight's holiday. She also said her father had died ten years before and then a few minutes later said she was worried because she thought she would never see her parents again. In the ward she was generally inactive but she would pilfer when possible. She smiled foolishly when asked questions and said "I don't know". On 1 January, 1956, she escaped from hospital but on 6 January, 1956, she was seen looking in shop windows in Newcastle by two nurses off duty and when reported to the police she came back to hospital willingly. When asked why she ran away she said "My mother was not very well", but this was a palpable lie as she did not go home or bother to contact her mother in any way. She hoarded useless rubbish of all kinds. At night she went to bed normally and when last seen by the nursing staff would appear to be settled down comfortably in bed, but in the morning she was usually found sleeping under the bed. When asked why she did this she smiled foolishly and replied "I don't know, Hinnny". During the day she usually sat behind a door on a lavatory seat and carried an empty cup in her hand, and persistently tried to scrounge fluid to drink. On tests of formal thought disorder she showed alolia.

Four patients were classified as depressive (eccentric) hebephrenics. The age of onset ranged from 16 to 47 years. However, in two cases where the age on admission was 47 and 34 years there was insufficient evidence of the age of onset and the ages on admission had to be accepted as ages of onset. Accepting these ages of onset the average age of onset was 30.75 years. Two of these patients had family histories of mental illness. One had a mother who was stated in the case notes to be insane but no further information was available. The other patient had a brother who died of Cerebral Arteriosclerosis in a mental hospital. However, one of the other patients appeared to have come from another part of England and would give no information about her family or herself.

B.F.H. is an example of this group. She was admitted to Northumberland County Mental Hospital on 28 January, 1916, when aged 19 with a history of abnormal behaviour since the age of fifteen. According to the certificate she accused her sister and father of insulting her and said she heard noises in the head. On 19 February, 1921, she was admitted to Newcastle City Mental Hospital when the notes state "Childish contented, says she was sent away because she lost her memory". Later in 1921 she is described as "Very childish. Has occasional fits of excitement." The notes about childishness continue until 1932 when in addition the notes state "At times ideas are exalted. Excitable and occasionally violent and is troublesome." In 1935 she is reported as "easily excited. Admits her temper is bad. Also hypochondriacal." In 1934 notes say "liable to manic depressive attacks". In 1947 a note reads "Labile, childish in outlook and conversation. Easily becomes excited." In 1956 she is reported as "Very agitated. Sometimes delusions. I have no neck, etc." When seen in 1955 and 1956 she was depressed and hypochondriacal. The depression varied in severity and appeared to be shallow. She had fantastic hypochondriasis which she brought forward monotonously and demanded treatment for. Her most persistent hypochondriacal delusion was that her bones had turned to jelly. At times her hypochondriasis was more vague and general. She showed marked alolia.

Four patients were allotted to the group of apathetic hebephrenia. These patients showed marked apathy punctuated by states of irritable excitement. The ages of onset ranged from 14 to 24 years with an average age of 18.5 years. Only one patient had a family history of mental illness. Her sister was classified as suffering from silly hebephrenia. One patient had had a pre-frontal leucotomy and this abolished her attacks of excitement.

E.McE. is a good example of this illness. She was first admitted to Gateshead Mental Hospital, Stannington, on 13 June, 1925, when 16 years old. There is no family history of mental illness. The admission note records "This girl is very depressed and quite demented. She never speaks or answers questions." On 15 June, 1925, there is a note "She is dull and stupid, takes no interest in her environment, emotional at times and weeps like a child." She improved and on 3 January, 1926, there is a note "Bright and cheerful. Mental condition completely satisfactory." On 3 May, 1934, she was admitted on certificate to the Newcastle City Mental Hospital. The admission notes record "Answers questions after a long time with an inaudible reply. Silly smile when addressed. Sits with head down. Dull, apathetic, wet and

dirty. Stuporous. Excited and agitated for a short while." On 30 June, 1934 she was transferred to Gateshead Mental Hospital. Here she was usually dull and apathetic but sometimes noisy and excited. On 13 October, 1934 there is a note "Excited and impulsive. Strikes out at other patients. Jumps out of bed and strikes the first patient within reach." On 30 May, 1935 she is described as dull and apathetic but impulsive at times. On 28 July, 1935 she was discharged and since this time she has been re-admitted six times to St. Nicholas Hospital. She was finally admitted to St. Nicholas Hospital under Section 20 on 24 November, 1944 and then certified. She has had phases when she was apparently depressed, irritable and impulsive and between the said phases she is dull and apathetic. The outstanding feature has been her apathy and this in conjunction with her irritable excited states allows the diagnosis of apathetic hebephrenia.

Eight patients were classified as autistic hebephrenias. The age of onset ranged from 21 to 50 years but clustered around the late twenties. In two patients with ages of onset at 45 and 50 years, the history of the illness was vague and the onset could have been earlier. Only one patient had a family history of mental illness in that her grandfather had been an inpatient in a mental hospital. A good example of this syndrome is M.F.

She was admitted to St. Nicholas Hospital on 29 September, 1925 when aged 26. She was discharged on 17 January, 1938 and re-admitted on 17 October, 1938. The notes on the earlier admission were not available. On her admission on 17 October, 1938 she complained of hallucinatory smells and said that the ward was full of gas and poisonous fumes. She said that there was nothing but sexual intercourse going on in the ward. She objected to answering questions. She asserted that her parents were not her real parents. She was treated with Cardiazol in 1938 and had 21 convulsions. After this she was described as pre-occupied, sullen and morose. In 1940 the notes state "She talks of a deceptive man from Brazil and his dirty daughter from Brazil in here". In 1942 she said her name was not M. She was always resentful of all attention, solitary and turned away from all contact. On 23 January, 1949 she struck a nurse on the head with a chamber pot and would give no reason for this. She tended to pay no attention to what was said to her. She usually muttered in a low voice and it was impossible to understand what she said. When interviewed by me she often refused to answer and on two occasions walked out of the room after a few minutes. On one occasion she said "I am sick of talking. Can I get out of here?" She talked in short jerky sentences and would give correct answers to questions but her diction was poor and she appeared to have thought disorder. If one picked out what appeared to be the correct answer from the unintelligible remainder of her speech and repeated it to her she would deny she had said such a thing. She whispered to herself all the time but she never was observed talking loudly to voices. Her autistic attitude with blunted emotional expression was outstanding. On tests of thought disorder she showed agrammatism and *Vorbeireden*.

When one considers the hebephrenias in the Kleist-Leonhard scheme the question arises whether this is a homogeneous group. The silly and depressive hebephrenias so well described by Leonhard (Leonhard, 1936) are certainly syndromes in which affect is devastated and the behaviour seems determined by this affective change. In Kleist's series (Kleist *et al.*, 1951) the apathetic hebephrenias are described as having severe excitements and in three cases there were furious impulsive actions. One wonders whether the group does not really belong to the catatonias. The autistic hebephrenics show an aversion to all approaches and deny all symptoms of illness. They could be considered as paranoid schizophrenics with aversion based on auditory hallucinations and persecutory delusions.

In this series the age of onset of silly hebephrenias was below 29 years. There is little doubt that it is an illness of early adult life or adolescence. The presence of the silly affectless behaviour could be related to the age of onset. It could be regarded as an accentuation of a normal variation of emotional behaviour.

The four cases of depressive hebephrenias in this group had ages of onset fairly far apart. This may be an artefact due to lack of precise information about the age of onset. It is not possible on the information available to relate

the depressive features to pre-psychotic abnormalities of mood or bodily pre-occupation.

DISCUSSION

These findings differ to some extent from those of Kleist and Leonhard. Although this sample was probably a reasonable selection of the female chronic schizophrenia population of a typical English mental hospital, this can always be doubted. The adequacy of the investigation can also be questioned. Nearly all the patients were seen on three different occasions and their behaviour was discussed with various members of the nursing and medical staff. However, more prolonged personal investigation might have revealed other clinical features. The objection to accepting descriptions from other observers is that if they do not know what to look for then they will not find all the signs which are present. This particularly applies to catatonia where careful descriptions of motor phenomena are difficult, and any description in Britain tends to be done in a stereotyped jargon in which the same word may cover many different phenomena. Thus the word negativism is used very loosely by most British psychiatrists whereas it has a very precise meaning to the Frankfort workers.

The other difficulty encountered was the adequacy of the clinical notes. Thus no consistent information could be obtained about the patient's previous personality, intelligence, school record or work record. Thus no estimation of cultural or social pathoplastic features could be made.*

The value of such a complicated classification as that of Kleist and Leonhard has been doubted. It appears to be valuable from several points of view. As it is based on careful clinical observation it increases the observer's clinical knowledge and powers of description. It also allows the cases seen by different observers to be compared and understood. Thus if an average British investigator designates a case as a paranoid schizophrenic no clear picture is brought to the reader's mind. If, however, a Frankfort worker designates a case as a combined incoherent-fantastic schizophrenic the informed reader will have a fairly clear idea of the symptomatology.

Probably the greatest importance of this scheme is its use in research into schizophrenia. If this scheme of classification is valid, as I believe it is, then it must bear some relation to the essential cause of schizophrenia. Kleist and Leonhard believe that the typical forms of schizophrenia are neurological system illnesses and the combined forms are combinations of such system illnesses. They regard the extensive atypical forms as extending beyond two systems and being caused by a disorder of the nervous system which is enhanced (possibly in a phasic manner) by a cause outside the nervous system, possibly an endocrine disorder.

Alternatively the classification could be explained by assuming one or more basic schizophrenic processes which are modified by pathoplastic factors. These pathoplastic factors could be different modes of functioning of the nervous system which in normal individuals account for some personality differences. Some of these neurophysiological differences might not yet have been observed in normals and could possibly only be elicited by specific techniques but would still modify considerably the expression of the schizophrenic basic process. Thus one might account for the different paranoid

* I would not like this to be considered as a criticism of the medical officers, past and present, of St. Nicholas Hospital. On British mental hospital standards the clinical notes of that hospital are among the best that I have seen.

schizophrenias, or the different confused schizophrenias on the basis of different strengths of the basic schizophrenic disorder and different types of imagery, and different intensities of auditory hallucinosis.

If pathoplastic factors could be isolated by using this scheme of classification then it might be possible to discover the basic neurophysiological disorder of schizophrenia.

In the past psychological and physiological investigations have assumed that all schizophrenics are alike and attempts have been made to understand the illness on the basis of one central psychological or physiological factor. It seems likely that a careful investigation of the psychological and physiological causes of the differences between schizophrenics could finally lead to a recognition of common causal factors in schizophrenia. The Kleist-Leonhard scheme of classification of schizophrenia provides a framework within which such an investigation could be carried out.

SUMMARY

All but two of a series of 107 chronic female schizophrenics, with a duration of at least ten years, were found to be classifiable according to the Kleist-Leonhard scheme of classification of schizophrenia. The distribution of these cases among the various subforms is given. Illustrative case histories of the different subforms are given. The possible pathoplastic factors in some groups of schizophrenia are discussed. The value of the scheme for further research into the causation of schizophrenia is discussed.

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NEUROSIS AND EXPERIMENTAL PSYCHOLOGY*

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GENETIC factors have been shown to play an important role in the development of neurosis. These factors, however, determine the vulnerability of an individual, the degree of his predisposition for neurotic breakdown, while the available evidence indicates that the actual behavioural disturbance results from the imposition of some form of environmental stress on the individual concerned. The probability that any particular individual will develop a neurosis is partly determined by his genetic constitution and partly by the degree of stress to which he is subjected.

Learning may be defined as the modification of an organism's behaviour resulting from its interaction with a changing environment and, in any learning process, the nature of the behavioural change will be partly determined by the genetic constitution of the organism. Therefore, in a real sense, a neurotic breakdown may be said to be learned, and recent experimental work linking learning theory and personality dynamics is extremely relevant to theories concerning the aetiology of neurosis. An aetiological theory expressed in terms of learning theory would provide a link with the general body of psychological knowledge and the possibility of validation in laboratory situations.

No one has stated an inclusive theory of this type in formal terms but the work of Mowrer (1950), Dollard and Miller (1950), Wolpe (1952) and others clearly indicates the main lines it would follow. These workers agree with Freud in allocating to anxiety a central role in the maintenance of neurosis but, to them, anxiety is conceived of as a conditioned fear reaction, and as such, will include affective and respondent components and will possess aversive drive properties. In its role as an acquired drive, anxiety will energize and, by its reduction, reinforce various aversive instrumental responses of the organism. As a powerful and massive response involving central, skeletal, autonomic and hormonal reaction systems, anxiety will disrupt the ongoing reactions of the organism.

The mediating role of anxiety is best illustrated by a consideration of traumatic avoidance learning in animals (Solomon and Brush, 1956). If an animal, confined in a suitable apparatus, is presented with a neutral stimulus followed by electric shock, a massive pain-fear response involving central, neuro-endocrine and autonomic reactions ensues and the animal makes various aversive skeletal responses, certain of which will, if the apparatus is appropriately constructed, terminate the shock and may therefore be described as escape responses. If, after one or several such trials, the animal is replaced in the apparatus, indications of fear are evident in response to the conditioned stimulus and before the onset of shock. This indicates that the fear-reaction has become conditioned to the previously neutral stimulus: such conditioned fear is described as anxiety. Furthermore, the previous instrumental aversive

* This article is based upon a paper presented to the British Psychological Society at Manchester in April, 1956.

responses are now made to the conditioned stimulus and escape from shock, gradually or suddenly, becomes avoidance of shock. Avoidance responses developed in this way, unlike the more usual instrumental responses of learning experiments, may be maintained throughout hundreds of extinction trials without the re-administration of shock. According to the mediation hypothesis this derives from the fact that reinforcement does in fact occur owing to a reduction in anxiety by escape from the conditioned stimulus.

To explain the failure of extinction of the conditioned anxiety response itself Solomon and Wynne (1954) have adduced two additional principles. The first, the principle of *anxiety conservation*, postulates that, by an early avoidance reaction, the conditioned stimulus is removed before the long latency anxiety response recruits to its full amplitude. If the avoidance response latency lengthens, anxiety develops fully, reinforcement of the instrumental response occurs and the latency again decreases. Secondly, they postulate that severe trauma causes irreversible physiological changes which bring about a *partial irreversibility* of conditioned anxiety responses to such traumata. This principle seems less satisfactory than the former and more economic explanations may be possible.

The animals in traumatic avoidance learning experiments do not usually develop behaviour which could be described as *experimental neurosis*. This is to be expected because the escape and avoidance responses are successful and the conditioned anxiety is playing an adaptive mediating role. Wolpe (1952) and others have shown that, when animals are subjected to traumatic shock from which they are unable to escape, marked behavioural disturbances occur, which may be claimed to merit the title "neurotic" in that they are unadaptive and persistent through a considerable period of time outside the experimental situation. These represent various persistent autonomic and skeletal responses which originally occurred in response to the shock and which markedly increase when stimuli associated with the shock situation are presented to the animal. Mowrer and Viek (1948), by means of an ingenious experiment, have shown that such abnormal responses in animals are not a function of the increased duration of shock when avoidance or escape is impossible but rather of the fact that no effective avoidance response may be established on the basis of anxiety reduction.

Physical pain of traumatic dimensions may be used as an extreme and prototypal example of environmental stress and, hence, the type of traumatic learning described may serve as a paradigm of the dynamics of anxiety mediated learning. In human development, of course, a large number of other aversive situations occur and many of these will derive from the socialization process, wherein individuals undergo a training in which socially unacceptable aspects of their behaviour, which are often strongly motivated, are punished by parents and the formal agents of society. The conflicts between powerful but incompatible response tendencies involved in such situations may be important in the generation of stress.

From such situations conditioned anxiety may well develop in a manner similar to that described for traumatic learning, and neurotic disturbance may ensue. Childhood may be a particularly vulnerable period in that it is a period of intense socialization, the undeveloped nervous system is more reactive and plastic, the possibilities of effective instrumental avoidance are less and anxiety may become attached to a wider range of conditioned stimuli owing to lack of learned perceptual discriminations and the absence of refined discrimination

through language development. This is not to say that a neurosis may not, however, develop at any period of life when new stresses appear.

On the basis of this theory the behavioural disturbance described as neurotic may be analysed into two types of response: the anxiety response itself which will have visceral, skeletal and central nervous components and various instrumental responses by which the conditioned stimuli are removed and hence the anxiety reduced. Both are learned behavioural responses and, as such, are symptoms of the neurosis: their division may parallel the Freudian distinction between the underlying neurosis and its symptoms.

With respect to the conditioned stimuli involved, these may derive from three sources: the punishing agent itself, for example the parent, when disturbances in relation to individuals in authority may result; the incidental environmental stimuli at the time of stress, when disturbances of the nature of phobias may be expected; the stimuli involved in the individual's own behaviour at the time of stress as, for example, when a neurotic subject fears that his actions may cause others harm. The latter type of conditioned stimulus is the least likely to be avoided successfully as it is, in a sense, self generated. As such, it may give rise to a type of neurosis particularly difficult to treat. This type of conditioned stimulation is the only one considered in their analyses by the most influential theorizers in this field: Dollard and Miller (1950) consider that, in these circumstances, the neurotic solution is to inhibit the offending behaviour, whereas Mowrer (1950) believes that the illicit response occurs but the consequences are avoided by various devices. In reaching these conclusions these writers fail to note that either adjustment, or maladjustment, is possible and rely on their apparently conflicting clinical experience. Whichever solution does in fact occur in the particular situation may be due to the circumstances of original reinforcement, that is according as to which of several responses made was effective in reducing anxiety. Alternatively, and more probably in the light of recent work, innate individual differences may largely determine the nature of the response.

Whatever the original conditioned stimuli, by the well-established process of stimulus generalization a range of stimuli will later evoke the neurotic behaviour, if these present themselves with considerable frequency the disturbance may well become chronic. Frequently the eliciting stimuli on various specific occasions may appear to an observer to be entirely dissimilar and extremely unlikely to occur on any single generalization continuum. This may well result from secondary generalization. Thus, if an animal, via the mediation of anxiety, has learned to avoid a black circle, it will, by generalization, also avoid a white circle. The avoidance of the white circle will itself be reinforced by anxiety reduction and, by a second generalization, a white square will now be avoided, though this stimulus has neither its squareness nor its whiteness in common with the original conditioned stimulus. Verbal links may also, in human beings, produce similar effects.

Many of these processes may be, in a sense, unconscious in that a number of experiments have shown that a response may be conditioned to a stimulus or class of stimuli of which the subject is consciously unaware. A dangerous topic of conversation may be avoided just as a signal of danger is avoided and this may also apply to symbolic responses or "thoughts", thus bringing the phenomenon of "repression" within the same conceptual scheme. From this point of view, however, the unconscious does not acquire any special "dynamic" attributes.

Individual differences will greatly affect the course of events in any individual case. Thus Lacey *et al.* (1953) have shown that individuals display a reliable pattern of autonomic responses to any particular experimental stress situation and that this pattern varies from situation to situation and from person to person. Such differences would clearly be important in symptom formation. Individual differences of a psychological character, particularly those related to learning, and their relationship to neurotic symptomatology have been discussed in a recent paper by Eysenck (1955).

In terms of this type of theory, neuroses are behavioural abnormalities which include ideational response components but do not derive from some psychic entity lying behind the symptomatic manifestations. If this analysis is valid all treatment must be symptomatic: if all symptoms are removed so is the neurosis. It is true that, if only instrumental acts mediated by anxiety are removed, the aversive drive tendencies will remain and, from the theory, one would predict that new instrumental acts would be reinforced and become habitual. This may be the foundation of the clinical belief in symptom substitution. In these instances the elimination of the anxiety responses themselves is also necessary. Some instrumental acts are themselves, however, the stimuli from which the anxiety responses derive. In writer's cramp, for example, the origin of the tremor may have been the result of a transient emotional disturbance. The disturbed writing behaviour would, however, lead to embarrassment and stress and, therefore, generate increased anxiety which would become conditioned to the various stimuli associated with preparation for writing. Liversedge and Sylvester (1955) have described a method by which this condition was symptomatically treated in a group of patients with no evidence of symptom substitution. The same is true of certain other well-defined symptom entities such as nocturnal enuresis when treated symptomatically.

As to techniques of treatment, as modification of behaviour is the aim, the generalizations of learning theory should provide the necessary guidance. Indeed, from one point of view, treatment may be considered as a problem in eliminating specific behaviour as presented and without reference to the causes and circumstances of their development and, where there is a circumscribed symptomatic entity with clear-cut stimuli defined by the situation as in the examples quoted of writer's cramp and enuresis, this approach may be adequate. In other instances, however, various remote stimuli are of such importance that a knowledge of the history of the condition is important. In terms of the present type of theory the diagnostic evaluation of a patient will need to refer to such things as his degree of neuroticism, his position on the extraversion/introversion continuum, the nature and amount of stress to which he had been subjected, the conditioned stimuli which evoke the abnormal reactions and his characteristic emotional and instrumental responses to those stimuli and the stress situation itself.

Various experimental circumstances result in the decrement or elimination of responses. Each may provide an analogous therapeutic situation.

Punishment: Thorndike postulated a negative law of effect as well as a positive, and punishment is frequently used in everyday life to control disapproved behaviour. The work of Estes (1944) and others indicates, however, that punishment merely produces a temporary interference with ongoing behaviour and does not reduce the probability of its evocation on future occasions. In this connection we need to distinguish between punishment and "escape from punishment", a rewarding situation often confused with punishment. Even if effective, punishment would be an undesirable technique for

dealing with responses mediated by anxiety, as the punishment itself would produce further stress and anxiety, which would summate with that already present.

Removal of unconditioned and conditioned stimuli: In the absence of the relevant stimuli, potential responses are not evoked and, in the long term, a process of forgetting or decay of habit strength may occur. This is precisely the rationale of Meyerian treatment by environmental manipulation, illustrated by the extensive social and educational modification carried out in child guidance clinics.

Satiation: Massed evocation of a response without reinforcement results in extinction of that response. After an interval, the response reappears owing to the phenomenon of reminiscence. Repeated massed extinction trials ultimately eliminate the response. This technique was adapted to the treatment of tics by Dunlap (1932), but his work has been largely ignored. Recently the writer has collaborated with Yates (1957) in attempting to eliminate a set of multiple tics in a young woman by this method. These tics are allied to respiratory movements and appear to have started following traumatic experiences while undergoing anaesthesia. During treatment sessions the patient voluntarily reproduces the tics as accurately as possible, and over prolonged periods of massed practice. Predictions on the basis of learning theory have been verified in this situation, but it has required considerable experimentation to establish the optimal conditions for the development of conditioned inhibition. At the present time the patient's voluntary responses have been much affected by inhibition, and there is a concomitant decline in her involuntary tics and a general improvement in her clinical condition. The questions still remain as to whether her tics will entirely disappear before or when conditioned inhibition of the voluntary responses is complete, and whether their disappearance, if it occurs, will still leave her with anxiety symptoms.

The modification of expectations: Contemporary learning theories take into account the mediation of responses by goal expectancies built up from experience of the reinforcing situations. When expectations are not fulfilled, as when the reinforcement situation is altered, new expectancies are developed and new instrumental behaviour mediated. Thus it might be thought that if the neurotic individual was forced or persuaded to remain in feared situations or to carry out feared acts, he would discover that the feared consequences no longer occurred. He would, however, meet the painful consequences of the unchecked arousal of anxiety, a response which, as previously described, is extremely difficult to extinguish. When traumatically trained animals are forced to reality test in this way, little modification of avoidance responses occurs. This is consistent with the fact that a patient with a severe phobia or obsession is quite well aware that his anxieties are unrealistic.

The strengthening of incompatible reactions: If one response, incompatible with another, is conditioned to the same stimulus as the latter, and the new connection progressively strengthened, the probability of evocation of the old response is progressively decreased, and will ultimately reach zero. This is the most promising method of response decrement for clinical application because emotional responses incompatible with anxiety are available in addition to incompatible instrumental responses.

One form of incompatible response is the opposite of the one to be eliminated. Actual inhibition of a muscular response is an example of this and is the principle underlying the well-known conditioning treatment of enuresis (Mowrer and Mowrer, 1938; Davidson and Douglass, 1950). The enuretic

responds by urinating to the stimulus of a full bladder. During treatment the same stimulus, the full bladder, is linked with a waking stimulus, a bell operated electrically at the commencement of urination. Urination responses are spontaneously inhibited on waking, and this inhibition, by a conditioning process, ultimately occurs spontaneously without waking and without sounding the bell.

Other incompatible responses involve essentially different activities from the treated one, but in so far as the same response systems are involved the two responses cannot occur simultaneously. This is usually the case when emotional responses are modified. When, however, a strong anxiety mediated response is involved it is extremely difficult to initiate and sustain alternative behaviour. Even if this were possible, intense conflicts would ensue which might themselves be the source of additional anxiety. It is in these instances that use needs to be made of the generalization continuum in order to reduce the response potential of the undesirable response. At the same time stimulation and motivation associated with the alternative response is enhanced. In these circumstances the alternative reaction becomes prepotent and, owing to its own generalization gradient, the process can be repeated nearer the centre of activity of the response to be treated. By moving along the generalization gradient in this way, the alternative response ultimately becomes prepotent in all circumstances.

This technique has an experimental foundation in Wolpe's (1952) animal experiments, and is essentially the method advocated by Jersild and Holmes (1935) for treating children's fears. Thus a child, afraid of dogs, is given a puppy. The puppy is sufficiently unlike a grown dog to elicit the fear to a small degree only. The puppy's antics create pleasurable responses, and as the puppy grows into a dog this attitude spreads to all dogs. This was the rationale for the treatment of a case described fully elsewhere (Jones, 1956). A patient's abnormal frequency of micturition was treated symptomatically by a conditioned inhibition technique. This symptom disappeared, but the prospect of venturing into the street continued to evoke a strong anxiety reaction, in line with the theory advanced in this paper. These emotional symptoms responded to a graded re-education programme devised in terms of the generalization principle.

Another example, illustrating the separate treatment of somatic and emotional symptoms, concerns a male patient who exhibited a marked tremor, associated with anxiety, and particularly evident when the patient had to write in front of others, especially his superiors at work, where he was a pay clerk. The tremor also occurred when holding a cup or glass in public, and in other similar situations. At the time the patient was first seen, the tremor was almost chronic. As the main symptom was somewhat akin to writer's cramp, the conditioning treatment described by Liversedge and Sylvester (1955) was applied. This includes practice in inserting a stylus into the progressively smaller holes of a steadiness tester. Contact of the stylus with the edge of the hole completes a circuit which applies shock to the patient's free hand. At the beginning of treatment the tremor was very marked, and the patient was unable to place the stylus in the largest hole of the tester. A regular learning curve was produced, and at the end of this phase the patient's skill with this apparatus was well above that of an untrained normal person. There was also a general improvement in his condition.

Even so, on those occasions when he had to sign documents in the office of his superior his anxiety was intense and the tremor reappeared. The generalization technique was employed with considerable success. Each day the patient carried out a task involving writing behaviour in public, but where the social consequences of failure were not serious and withdrawal to privacy was

possible. Thus, for example, he might take a cheque to pay into his bank account and deliberately fail to endorse it. When the cashier pointed this out and requested his signature, the patient would be prepared and could, if overwhelmed with anxiety, retire to a writing booth. This case has been followed up for over a year without relapse.

Herzberg (1941, 1945) reports good results on a large group of patients with a technique involving graduated everyday tasks of a similar nature, but real life situations may not be necessary in all cases, at least in the early stages of treatment. Psychodrama may well be adapted to this theoretical formulation, and has been used in treating a child patient with an aggressive disorder. This type of psychodrama does not, however, like psychotherapeutically orientated psychodrama, involve the "acting out" of existing attitudes and responses, but their suppression by alternative incompatible responses from the earliest stages. Graded situations of this type may also be suggested in hypnotic states, a technique used by Wolpe (1954).

In chronic and widely generalized conditions, it may be extremely difficult to find a situation in which the anxiety response is sufficiently weak to be superseded. This is when hospitalization and drug treatment may be important. Thus Meyer (1957) rapidly and successfully treated a severely phobic and anxious patient on these lines, once she had settled in the hospital ward and felt relatively secure there, despite the fact that, at first, any emergence from the ward induced severe anxiety and psychosomatic symptoms.

Removal of "manipulanda": Another possible method of response decrement, essentially similar to the last, is to eliminate from the stimulus situation those aspects necessary for the performance of the undesired response, while the remainder of the stimulation remains intact. Thus, if a rat spends much time in a Skinner box, from which the bar is removed, earlier bar pressing responses will be eliminated (Hurwitz, 1955). Similarly, if the tremorous patient previously described was able to spend a considerable period each day with his employer, but without being required to write, a later demand for his signature might produce no marked effect.

Throughout the discussion of treatment the effects of individual differences, in terms of learning parameters, have been ignored. These may frequently be of importance. One patient (Meyer, 1957) was practically unconditionable in a laboratory test. Excitatory drugs were therefore used during the training period and then withdrawn, as, once learning had occurred, there was no reason to believe that his retention would be impaired. This procedure is justified in terms of animal experiments. Fairly obvious and rather mechanical applications of therapeutic principles have been stressed, but verbal interactions between therapist and patient may also be manipulated along these lines. The concept of the transference situation might well fit into this conceptual framework: the therapist will presumably lie somewhere on the stimulus generalization continuum derived from, say, the patient's father, and will, therefore, elicit responses learned in relation with the father. In so far as these are modified, the new responses generalize back to the father-son relationship. The present theory would, however, tend to indicate more active treatment than is usual in psychotherapy, and would focus attention on the fostering of new responses rather than on the "understanding" of old responses.

Finally it needs to be stated that, though the apparently successful treatment of a few cases has been described, there are at least two cases, treated on similar lines, which were not successful. It is not claimed that the remainder necessarily improved as a result of treatment, nor that retraining techniques are

clearly better than psychotherapeutic procedures. What is claimed is the possibility of devising treatments derived from the findings of general psychology. Large-scale controlled studies involving such treatments would be necessary to establish their efficacy. The reported results of workers like Wolpe and Herzberg encourage optimism as to the results of such an investigation.

SUMMARY

Neurotic behaviour is considered in the light of the concepts and findings of experimental psychology, and the possibility of an aetiological theory in these terms is examined. The implications of such a theory for the treatment of neurotic patients are indicated and examples of treatment from this point of view are quoted from clinical practice and the literature.

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PREVENTIVE PSYCHIATRY— IS THERE SUCH A THING?

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THE task of preventive psychiatry—if there is such a thing—can be expressed in a positive form as the promotion of mental health; but mental health can be defined in two ways: as the absence of recognizable mental illness or, in more grandiose terms, as the realization to the fullest possible extent of one's personal potentialities. The latter concept has been denounced by some authorities as representing much too wide an extension of psychiatry: that it continues to be prominent in discussions of mental health programmes and the like is due to the insistence of laymen perhaps even more than of psychiatrists. It appears that in the Western World people are increasingly turning to psychiatrists for the answer to problems which once would have been the concern of priests or politicians. That they do so is a psychiatric problem in itself; that they will continue to do so in increasing numbers seems very likely—so we had better have our answers ready, even if they are only disclaimers of problems which do not concern us.

The trouble is, of course, that it is so hard to draw the line between plain unhappiness and neurotic illness. To an increasing degree, it seems, it is coming to be believed—especially in the American urban middle classes—that to be unhappy is itself a mark of illness. Over thirty years ago Aldous Huxley wrote a pessimistic fantasy entitled “Brave New World”. In the synthetic, de-individualized society which he envisaged, all citizens were conditioned to share identical tastes and prejudices, and to live in a perpetual state of unreflective euphoria. If ever they were threatened with anxiety or distress, they would swallow a gramme of “Soma”, and euphoria would be restored.

Today, in America, this prophecy is becoming realized. The sales of “tranquillizers” amount to many millions of pounds a year. Readers of the evening papers may already be aware that Hollywood is coming to be known as “Miltown, U.S.A.”; that James Mason's director fortifies himself with an ataraxic before interviewing him—only to discover that Mr. Mason does the same. A significant proportion of New Yorkers take sleeping tablets every night—because in this highly conformist society it excites anxiety to lie awake when everyone else is sleeping. (By a dramatic irony we find Huxley himself, a generation later, extolling mescaline as a—for him—new-found elixir, giving access to experiences of transcendental value.) In Britain, too, tranquillizers are bidding fair to displace phenobarbitone as the commonest drug in use.

These are attempts, after a fashion, at preventive psychiatry. The scale on which they are practised reminds us of the need to examine our own psychiatric resources.

I do not propose to devote much space to considering this wider meaning of mental health; except to point out that “divine discontent” (Kingsley, 1874) is an age-old attribute of mankind. It may even be regarded as an agency of evolutionary progress. In every society there have been satisfied conformists

and dissatisfied rebels. Although the antithesis is by no means absolute, one can say that where dissatisfaction was centred on the material conditions of life, its expression has usually been political; where it was a protest against spiritual impoverishment its inspiration has been religious. We read in St. John: "I am come that they might have life, and that they might have it more abundantly"; and many other religions promise a similar enrichment of the personality.

Psycho-analysts (and perhaps especially analysands) do not like to be described as adhering to a faith: and yet in this respect they have inherited a religious tradition. How many people are there today who would be willing to spend five hours a week, at two or three guineas a time, in conference with a trusted clergyman, in the hope of bettering their wayward natures? Analysands are the *dévots* of our age.*

Historians tell us that unrest and non-conformism are always most evident in times of rapid social change. We are living through such a period now, but with a difference: never before has the social ferment been on a world-wide scale. Peoples' material discontents have been sharpened by the awareness that in the near future it will be technically possible for the first time on earth on the one hand to eradicate hunger and want, and on the other hand to exterminate the human race. There is some evidence that modern civilization, while conquering the major infective diseases, is giving rise to new pandemics of psychosomatic disorders in whose treatment and prophylaxis the psychiatrist is increasingly involved (Halliday, 1948). It is certainly the case that the educated public is showing a new awareness of the avoidable distress occasioned by minor emotional disorders, and by faulty human relationships. In consequence, psychiatrists find their advice sought in spheres with which they may be unfamiliar—such as education (of parents as well as children), vocational guidance, marriage guidance, personnel selection and management, problems of delinquency and crime, etc. One alternative is to abdicate from these responsibilities with a plea of incompetence; but it may be more realistic for psychiatrists to recognize these pressing demands, and to see that some members of the profession are suitably trained to meet them.

To return to the more strictly medical aspects of psychiatry: no one would question that during the present half-century there have been great changes in our practice and in the type of case we treat. When Sir David Henderson (my first teacher in psychiatry) began work in the Royal Edinburgh Hospital in the early years of the century, every third bed in the infirmary ward was occupied by a general paralytic; and there were many cases of gross alcoholic dementia. Both of these conditions have now dwindled to very small numbers indeed. Prevention has taken effect—but can psychiatry claim the credit? These are instances, rather, of the success of strictly medical and social interventions respectively.

MEDICAL PREVENTIVE MEASURES

Preventive medicine has made enormous advances in the last fifty years. Its mastery of syphilis has been the biggest direct contribution to psychiatry. Others which come to mind are the discoveries of effective preventive measures against pellagra and cretinism. More recently, obstetrics has also come into the picture. It has been shown that some cases of mental deficiency can be attributed to brain injury resulting from complications of pregnancy and

* I speak as an *ex-dévol*, but by no means an apostate.

childbirth: among these are eclampsia, asphyxia of the child and physical trauma.

Another, perhaps less obvious, relationship between obstetric care and the prophylaxis of psychiatric disorders was brought out in a recent study from Johns Hopkins University, reviewing the birth histories of 1,151 behaviour problem children and 902 matched classmates, from public schools in Baltimore (Rogers, Lilienfeld and Pasamanick, 1955). Abnormalities of the pre-natal and paranatal periods were found to be significantly associated with behaviour disorders in children. In the authors' view this lent support to the hypothesis that in some cases minimal brain damage may have occurred: they provisionally class them as belonging to the "continuum of reproductive casualty" (which includes at the other extreme foetal and neonatal mortality, cerebral palsy and epilepsy). The discovery that a specific infection (German measles) caught by a mother at a crucial stage in the development of the foetus may lead to brain damage as well as to deafness or blindness in the child has opened up the possibility of further preventive measures through improved obstetric care.

In one respect, the rapid advances in medicine have proved an embarrassment to psychiatry. Thanks to antibiotics and chemotherapy, everyone is living longer, including chronic psychotics and imbeciles. Many more people than before live long enough to run the risk of suffering from cerebral arteriosclerosis or senile psychoses—and this has resulted in mounting admission rates to mental hospitals.

SOCIAL PREVENTIVE MEASURES

The reduction in severe alcoholism in this country can confidently be ascribed to taxation, which has made it so expensive to drink really heavily. Similarly, legal sanctions have cut down the amount of drug addiction. Other preventive measures of a social kind are the successive modifications of the laws governing the treatment of the mentally ill and the mentally defective: modifications which are still going on, but which have already done much to relieve the stigma of mental illness, and to facilitate early treatment. The National Health Service Act was itself an important preventive measure, because it removed the financial barrier to seeking early treatment, and accelerated the co-ordination of psychiatric treatment with that of the general hospital.

Anything which helps to overcome public dread of mental illness, and reluctance to seek expert advice, is a step in this direction: judicious propaganda, like the recent television series "The Hurt Mind" and current radio programmes on problems of mental health and mental deficiency, has some effect—but the most potent propaganda of all is the demonstration of successful therapy. Within the last ten years we have seen a dramatic change in public attitudes to tuberculosis—because, thanks to isoniazide, streptomycin and P.A.S., sufferers are seen to recover and to be able to lead normal lives. Similar results are accruing from the results of modern methods of treatment in psychiatry. If public attitudes are slower to change with regard to mental illness, this may be because our treatments are still seen to be only partly successful.

There are several instances in which medical and social agencies work together in order to prevent, or to minimize, psychiatric breakdown. In the armed forces, for example, prevention begins with the screening of recruits and of combat troops: at least, this was the intention, but so much uncertainty still attends psychiatric prognosis that the normal screening procedures had a

wide margin of error. In one remarkable study (Plesset, 1946) a divisional psychiatrist had an opportunity of observing how 138 soldiers, who had recently been brought to his attention with neurotic and psychopathic symptoms, fared in five months of combat experience. During this period, only three required to be evacuated on psychiatric grounds, whereas eight were awarded medals for meritorious service; at the end of the war, 120 were still on duty. Similar reports from psychiatrists in the German as well as the Allied armies have given many instances in which the performance under stress of neurotic individuals exceeded expectations (e.g. Kalinowsky, 1950; Hunt, Wittson and Hunt, 1952).

In recent wars, psychiatric casualty rates have been shown to fluctuate with unit morale, and also with the tide of battle; they generally rise in proportion to the rise in the rate of wounded and killed, but a notable exception is during a full retreat. For example, at one stage in the Korean war the United States forces were in retreat and the enemy were said to be practising atrocities on the captured prisoners: for a time there were very few psychiatric casualties indeed (Glass, 1953).

This recalls a view which Dr. C. P. Blacker has put forward: that there is no preventive of neurosis so strong as the experience of *shared* physical distress. It was repeatedly shown in Germany (Kalinowsky, 1950) that psychiatric cases were least common where populations were exposed to severe bombardments. This might be interpreted as a case of the greater evil driving out the lesser; but it also indicates the importance of group solidarity as a prophylactic against breakdown. Good morale in a unit, as competent officers came to learn, can be fostered: it demands contiguity, time to get to know one another, free channels of communication throughout the group, a sense of shared purpose, or at least of a common fate. Out of these emerge a readiness to help each other, and a confidence that help will be given to oneself in time of need.

These attributes of good morale are found in many closely integrated primitive communities: they are nowhere so conspicuously lacking as in the areas of social disorganization which can be found in large industrial cities. A succession of monographs, from the pioneer study in Chicago (Faris and Dunham, 1934) to quite recent investigations in London (Sainsbury, 1955) and Bristol (Hare, 1956) have shown that suicide, delinquency and some mental illnesses (particularly schizophrenia) occur at a higher rate in socially disorganized areas: but it is easier to diagnose this situation than to offer a remedy. Social services are the contemporary substitute for the "neighbourliness" of integrated communities. They are efficient in providing against destitution, but cannot annul the loss of personal contact. There is no sense of give and take in stamping one's insurance card, and drawing "benefits accrued"; often where the need is greatest the receipt of "national assistance" is felt as a disgrace.*

We have to face the fact that social circumstances may sometimes be so adverse that a normal personality adjustment becomes almost impossible: for example, in the 'thirties Professor Lewis studied a group of chronically unemployed men whose mental health was in question (Lewis, 1935). In several cases he concluded that their mental state was either caused or much aggravated by their unemployment—but it was little use to prescribe work at that time of economic depression.

*During a follow-up study of ex-mental hospital patients, in which the writer is currently engaged, it was found that one man made it his first task to pay back £300 which had been paid to his wife by the National Assistance Board. He felt that until he did so he could not remove the slur of being a pauper.

Fortunately, social exigencies are not always so hard to meet. During the war the same author and a social worker colleague analysed the social causes of admission to a mental hospital for the aged (Lewis and Goldsmidt, 1943). They concluded that the admission of many of these old people could have been avoided by timely social work. Since then Dr. Cosin at Oxford and Dr. Macmillan at Nottingham have shown in practice that this is so.

As we know, at this time several experiments are under way to test the advantages of community care for mental patients, on the Querido plan. I include these under the heading of social measures because what they prevent is not illness (though this may prove to be the best method of treatment in some cases) but the social fact of admission to hospital.

On the other hand society may be able to influence the amount of its psychiatric casualties through education. This is clearly shown in the case of mental defect. A number of recent studies (e.g. O'Connor and Tizard, 1956; Clarke and Clarke, 1953) have shown that in many cases the defect is not an insuperable barrier to learning; in some, intellectual maturation is delayed rather than absent. If appropriate measures of graduated social and elementary scholastic teaching are applied, many of these patients can be restored to a socially useful existence. This process is being accelerated at the present time by social pressures applied by Lord Goddard on the one hand and an inflamed public opinion on the other.

A more general question is whether education, in school and in the home, can influence the incidence of mental illnesses. This question is so closely linked with psychological theories about the aetiology of these illnesses that I propose to consider it in the next section of my paper.

PSYCHIATRIC PREVENTIVE MEASURES

So far, I have briefly discussed ways in which preventive medicine and social measures have contributed to the prophylaxis of psychiatric disorders: but are there any preventive measures which are clearly *psychiatric*? I suggest that there are—but not many.

Prevention can operate at three levels:

1. Where the essential aetiological factors of a disease are known, timely intervention may prevent their occurrence.
2. Effective treatment, applied early, may limit the development of illness.
3. Treatment of established illness can be designed to alleviate the disabilities which it entails.

Let me begin with the third level. One way of putting it is this: even if we cannot *cure* serious mental illnesses, are we doing all we can to prevent our patients from getting worse? There is a growing realization that the regime which used to prevail in all our mental hospitals—and which still prevails in some—had the effect of increasing patients' helplessness and regression. To a considerable extent, so-called "secondary dementia" is a hospital artefact: this is being proved today in many hospitals in which active social treatment is both arresting and reversing the process in long-stay patients.

Another important field of ameliorative prevention, which is as yet inadequately developed in this country, consists of the supportive follow-up of discharged patients. To be really effective, this calls for the co-ordination of psychiatric and P.S.W. efforts. There is an interesting report (Stevenson and Geoghan, 1951) of a five-year experiment in psychiatric prophylaxis, in

which the authors placed reliance upon regular out-patient E.C.T. as a prevention against recurrent manic-depressive illness. They found that patients were strangely reluctant to undergo shock treatment once a month for five years, even when they felt perfectly well; and they found some cases in which the prophylaxis did not work. For these they recommended prophylactic leucotomy! In a number of cases it became plain that the illness was related to adverse home circumstances, and the help of social workers was invoked: to this extent, at least, the experiment resembled the sort of co-ordinated follow-up which I have mentioned above, and which is already an established part of psychiatric practice in some parts of Holland.

Secondly, early treatment: our profession takes pride in the many new empirical treatments which have been introduced in the last twenty years, and public confidence in the value of these treatments has been indicated by the great increase in the numbers of patients who come voluntarily for treatment at an early stage of their illness. In some instances (such as the use of E.C.T. for involutional depression) the beneficial results are unmistakable; there is little doubt, again, that in the last war energetic measures of treatment in the forward areas proved effective in preventing much chronic psychiatric disability (Gillespie, 1942; Appel and Beebe, 1946). In other cases (such as the sweeping claims which are made for each new tranquillizer as it appears) our evaluation has to be more cautious, because results attributed to these drugs, as in the past to leucotomy, may be due in part to more general influences, such as the "Hawthorne effect" of receiving special attention and increased social stimulation.

In this matter of the systematic assessment of the results of therapy, psychiatry is only now emerging from its neolithic stage. It is no longer respectable to publish reports based on subjective "clinical impressions"; but as soon as we try to be more rigorous in evaluating the outcome of treatment, we are confronted with the baffling complexity of factors which may influence the course of an illness.

If this is true in the comparatively simple case of assessing physical treatments of established illness, it applies with still greater force when we consider claims that it is possible to intervene in a specific way, so as to prevent or minimize the development of mental illness in persons exposed to risk.

This presupposes a clear-cut, accepted explanation of the way in which such illnesses are caused: but no such explanation exists. In his outstanding monograph on dementia praecox, Eugen Bleuler was quite candid about this. He wrote: "As yet we do not know of any real prophylaxis for this disease", and added, with a sort of desperation: "The avoidance of masturbation, of disappointments in love, of strain or frights are recommendations which can be made with a clear conscience because these are things which should be avoided under all circumstances" (Bleuler, 1950).

Even if we knew how to intervene to prevent schizophrenia, it might not be easy to know when to do so. In an early paper, Kasanin showed that when he investigated the school records of schizophrenic patients he found that the great majority had been regarded by their teachers as either model pupils, or as presenting no problems (Kasanin and Veo, 1932).

My own experience in child psychiatry was the obverse of this. In 1949 I worked for six months in a child clinic in New York, principally with a group of eight children aged about five. In accordance with the prevailing fashion, nearly all of these children (*and* their mothers) were diagnosed as schizophrenic. When I revisited New York 6½ years later I called at the clinic and asked for

news of them. In every case I inquired about, the child was now in a normal school class and getting on quite well.

We probably should all agree that inherited constitutional factors can predispose a person to some forms of illness: but except in rare cases, the amount of such predisposition remains conjectural, and the advice which we can reasonably give if we are consulted about marriage and procreation is correspondingly qualified.

On the other hand, there is wide divergence of opinion within psychiatry about the degree to which environmental factors contribute to mental illness, and about the manner and the stage of development at which they do so. Psycho-analysis represents in the clearest and most authoritative form the view that early psychological experiences play a vital part in aetiology, that their harmful results can be modified by psychotherapy, and that psycho-analytic theory can indicate ways in which the upbringing of children—in the home and in school—can be carried out so as to minimize the risk of later neurotic or psychotic breakdown.

Fenichel puts it in this way: neuroses are based on neurotic conflicts; neurotic conflicts are created in childhood between instinctual impulses and the fear of dangers associated in fantasy with yielding to these impulses. Everyone, in the process of growing up, finds his instinctual impulses confronted at times by limitations inherent in the reality situation. Faulty education consists in confronting the child with threats which are out of proportion to the real danger, so that he takes fright and his impulses are not merely restrained but repressed—to the detriment of his emotional health in later years (Fenichel, 1945).

Psycho-analytic theory has inspired a great deal of the practice of child guidance clinics; and it has also had a considerable indirect influence upon education and upon the way modern parents treat their children. Fenichel gave a few practical hints for child-rearing derived from his theory of neurosis: avoid premature excitation of sexual feelings, and avoid exaggerated condemnation if these occur; delay toilet training until the child is physiologically prepared to respond, and do not force it; anticipate emotional trauma, such as the birth of a sibling, the absence of its mother, or an operation so that the child will not meet it unprepared; in matters of discipline "it is better to understand the child's needs than to use rigid disciplinary patterns".

To a considerable extent all of these maxims have already been accepted by enlightened parents and teachers. It is also becoming widely realized that when one member of a family is emotionally disturbed, another may be in need of treatment.

Yet it has to be admitted that there is *no* evidence that the work of child guidance with patients or with their parents has made any significant difference to the attack rates of neuroses or psychoses in any community. These therapists are inspired by convictions which are based on faith rather than certainty. Personally, I think no less of them for that, provided that they remain willing to re-examine their premises in the light of objective evidence whenever this becomes possible. To refrain from action if there is a possibility that we may do the patient harm is a reasonable precaution, but to continue to refrain until we are absolutely convinced of its rational justification is to succumb to *folie de doute*.

A celebrated example of the danger of uncritical acceptance of an aetiological theory is given by the history of what has come to be known as the "Bowlby hypothesis" concerning the far-reaching harmful consequences of

maternal deprivation during a child's first three years. Dr. Bowlby's book *Maternal Care and Mental Health*, which was published by the World Health Organization in 1951, contained descriptions of cases and cited evidence from other workers which provided very strong support for his contention. The publication of a "Pelican" version of his report made his views widely known: with important social results. One result has been that many children's hospitals and obstetric units have modified their rules in a way which must make the hospital experience certainly more enjoyable and probably more beneficial for their patients; but another result has been to spread alarm and despondency among mothers, and among those responsible for orphanages and similar institutions. Had the "Bowlby hypothesis" been firmly established, this might have done no harm, but in fact, as he himself very honestly admits, in the last five years a number of studies have clearly shown that maternal deprivation is not always accompanied by such dire consequences as many people had been led to believe (Bowlby *et al.*, 1956). Even Margaret Mead, at first an enthusiastic protagonist, now sees its uncritical acceptance as "a new and subtle form of anti-feminism in which men—under the guise of exalting the importance of maternity—are tying women more tightly to their children than has been thought necessary since the invention of bottle feeding and baby carriages" (Mead, 1954). Dr. Bowlby is now engaged in refining the concept, and in analysing additional factors which may mitigate the effect of maternal deprivation.

Before concluding this brief review of preventive psychiatry, I should like to point out that psycho-analysis gives especial prominence to one element in the treatment of mental disorder which has long been part of good psychiatric practice—which is, indeed, as old as the Hippocratic oath: namely the relationship of mutual respect between doctor and patient, the readiness to help and (when volition is not lost) the readiness to accept help. There is no statistical proof that sincerity and humanity in one's dealings with people in need will influence their future mental health; but this is an act of faith which I, and many others, have no difficulty in making.

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A CENSUS OF PSYCHIATRIC CASES IN TWO CONTRASTING COMMUNITIES

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INTRODUCTION

DURING recent years members of the Medical Research Council Pneumoconiosis Research Unit have carried out a number of studies of the prevalence and incidence of common diseases in two populations in South Wales and attempted to relate differences in morbidity to known differences in social and environmental factors (Cochrane and Miall, 1956). The communities studied are the Rhondda Fach, a mining valley with a population in 1951 of 19,722 (aged over 15 years) living in a contiguous series of eight small towns, and a number of parishes in the Vale of Glamorgan, approximately ten miles from the Rhondda Fach, in which the population of 4,621 (aged over 15 years) is spread over a relatively large agricultural area. These will be referred to as the Rhondda and the Vale. They were originally chosen to provide a complete mining community (in which the prevalence of diseases of the lungs in miners and ex-miners could be estimated) and a contrasting, predominantly rural, population respectively.

It was found in successive investigations that random sampling of these populations provided valuable information about the epidemiology not only of diseases of the chest (Cochrane *et al.*, 1952, 1955, 1956) but also of coronary heart disease (Thomas, Côtés and Higgins, 1956), hypertension (Miall and Oldham, 1955) and rheumatoid arthritis (Miall, 1955).

Through the co-operation of the Pneumoconiosis Research Unit the opportunity was presented to extend these epidemiological studies to measure certain indices of psychiatric morbidity in the two areas during a period of five and a half years. Where differences of "ascertained prevalence" of psychiatric disorders were observed, an attempt was made to relate these findings to sociological differences between the populations at risk, as a first step towards establishing the degree to which socio-environmental factors contribute to the recognition and treatment (if not to the aetiology) of certain manifestations of mental illness. It is necessary therefore to summarize their respective social features.

SIMILARITIES AND CONTRASTS IN THE COMMUNITIES

The Rhondda and the Vale are not far apart. Both lie in the county of Glamorgan. Information was not obtained about the relative incomes per head, but it was found that overcrowding (persons/rooms) was not significantly different in the two areas. Their inhabitants are of mixed descent, and all speak English; only a few members of either community also speak Welsh, and these

are of the older generation. The two populations are served by the same two mental hospitals and four psychiatric out-patient clinics. None of the four hospitals providing psychiatric treatment lay within the two areas but all were within easy reach. For most individuals there was a clinic or mental hospital within a 15-mile journey, and for many within a shorter distance.

In addition to the original criterion of the presence of coal-mining in one and its absence from the other area, the communities differed in the following respects:

(i) *Density of population.* The larger Rhondda population is concentrated in a relatively much smaller area. The Vale has a very low population density. As a result, the appearance of the Rhondda is predominantly industrial (even the adjacent hills being surmounted by slag-tips) whereas the Vale presents an unspoilt rural landscape.

(ii) *Occupational distributions.* The Pneumoconiosis Unit's survey in 1953 showed that the Rhondda has a greater homogeneity in male occupations, 70 per cent. of the male population over 15 being miners or ex-miners* while the major industry in the Vale, farming, claims only 36 per cent. of the men over 15. Comparison of the two populations in terms of social class (using the Registrar General's five-fold classification by occupations) shows a relatively greater representation of the upper occupational classes in the Vale. The percentage distribution of men over 15 in the Vale is: Classes I and II (professional) 10 per cent.; Class III (skilled) 71 per cent.; Class IV (partly skilled) 12 per cent.; Class V (unskilled) 7 per cent. The distribution in the Rhondda is: Classes I and II—8 per cent.; Class III—52 per cent.; Class IV—28 per cent.; Class V—12 per cent.

(iii) *Public transport.* The small towns which comprise the Rhondda are strung along a single main road which runs down from the head of the valley, leading to the city of Cardiff; bus services are frequent and easy of access. The scattered farms and small villages of the Vale are served by relatively infrequent and less readily accessible country bus services.

(iv) *Changes in population.* The Vale has been farmed for many centuries; its Royal Borough of Cowbridge (population 1,000) has a charter dating from the 13th century. In contrast, until the discovery of its coal fields a little over a hundred years ago, the Rhondda was a bleak valley inhabited only by a few shepherds and their flocks; as each new pit was dug it attracted workmen from Ireland, North Wales, Somerset and the Midlands. The peak population increase was reached in the first decades of the present century. During the depression of the nineteen-thirties many emigrated from the Rhondda. During and since the second World War improved conditions in the mines and the provision of alternative light industry have arrested the flow of emigration. In the Vale there are numerous instances of relatively prosperous incomers buying residential property, or farms, within the last twenty years. The size of these population shifts has not been established.

(v) *Ascribed characteristics of the two populations.* In addition to the above more or less objectively demonstrable differences, the populations were regarded by the consensus of local opinion (in which they themselves shared) as exhibiting quite markedly contrasting social traits. People from the mining valley were described as being "rougher", more prone to drunkenness, to violence, to petty crime and to immorality, more talkative and more belligerent than those from the Vale, given to extremes of left-wing politics on the one hand and of chapel-

* Men are only defined as "ex-miners" if they have been employed at least six months in work involving underground activities.

dominated puritanism on the other. They were known to have enjoyed relative prosperity in the last fifteen years, but this was generally said only to have increased their consumption of alcohol, and with it their emotional instability. In contrast, the farmers and other country dwellers in the Vale were regarded as relatively placid, stolid people, slow-moving and law-abiding, who might have their share of idiosyncrasies but were accustomed to keep themselves to themselves and to avoid the public display of emotions.

Although these two antitheses have been simplified to the point of caricature, they do represent patterns of behaviour ascribed to these two communities by local verbal tradition. In short, the Rhondda was said to exhibit many of the attributes of social life in an industrial slum, whereas the Vale appeared to be an unexceptional rural community. Since the former presented a picture of relatively greater "social disorganization" (Faris and Dunham, 1939) or of *anomie* (Durkheim, 1930) it was expected that rates for crime, suicide and psychiatric illness would, as postulated by these authors, be higher in the Rhondda community than in the Vale. As will be seen, not all of these expectations were fulfilled.

AIM AND METHODS OF PRESENT STUDY

An attempt has been made to ascertain the numbers of presenting cases of mental disorder in the two communities by counting all persons admitted to mental hospital, all those seen at psychiatric out-patient clinics, all cases of suicide and attempted suicide, and also the totals of certain indictable offences (sexual offences and cases of bodily assault) occurring in the two populations in the 5½-year period from 1 January, 1951 to 30 June, 1956. The number of patients in mental hospital at the end of this period was also determined. The age and sex distributions of each population and also certain details of male occupational histories were available from the Pneumoconiosis Research Unit. It was thus possible to establish psychiatric sickness rates for both men and women in relation to these two communities and also, within the larger Rhondda community, to compare the rates shown by miners and ex-miners with those in the remainder of the male population.

The study is based almost entirely on documentary evidence. For the counts of psychiatric in-patients and out-patients, and indictable offences, the study shows how many cases came to be formally recorded during the period under review. For cases of suicide and attempted suicide, evidence was also sought from general practitioners. No attempt was made to interview patients, nor to ascertain a "true count" of psychiatric cases in the populations.

Search was made of the records of all mental and general hospitals serving the area in order to complete the following counts:

(a) *Patients in mental hospital on 15 July, 1956.* (All other counts were of cases during the 5½-year period under review.)

(b) *Patients admitted as in-patients to mental hospital, psychiatric unit or observation ward in the 5½-year period.* To avoid overlooking patients who had sought private psychiatric treatment outside the area, inquiries were made at ten private hospitals, yielding in all two extra cases. General practitioners were also consulted about possible private mental hospital patients.

No attempt was made to assess the number of psychiatric cases (except for attempted suicides) in the general hospital population because of diagnostic difficulties.

(c) *Persons seen by psychiatrists in out-patient consultation in the 5½-year period* (including those seen in private consultation).

(d) *Cases of suicide.* As recorded in the records of the Regional Medical Officer of Health in the 5½-year period. Death certificates were scrutinized to confirm possible instances of suicide where an open verdict had been made.

(e) *Cases of attempted suicide in the 5½-year period.* The eighteen G.P.s and consultants who cover the area were consulted as well as the case records of the ten local general and cottage hospitals, paying particular attention to cases of laceration, asphyxia or poisoning: only those in which definite evidence of suicidal action was found were included.

(f) *Persons charged with specified offences in the 5½-year period* (crimes of violence and certain sexual offences believed on *a priori* grounds to be frequently associated with mental disorder) were obtained by our being given access to the record system at the headquarters of the County Constabulary. Only crimes committed within the specified areas of this study by members of the population were recorded.

In all the above counts only persons over the age of 15 at the time of treatment or occurrence were included.

The County Council records of ascertained mental defectives were examined but were not used in this study as they were declared to be far from complete. Only a few mental defectives actually admitted to mental hospitals have thus been included in our figures: those in special Mental Deficiency institutions have not been included.

RESULTS

(i) COMPARISON OF FINDINGS IN THE TWO POPULATIONS

To allow for age and sex differences between the two communities, the Rhondda figures, which relate to a population over four times larger than the other, have been corrected by standardizing them to the Vale figures in each 5-year age-sex group. The standardized figures can then be compared with actual totals for the Vale to give a comparison between rates in the two areas. In the following account standardized figures for the Rhondda are used unless otherwise specifically stated.

The results for all counts are shown in Table I: the figures refer to persons and not events except that for admissions in the 5½-year period events are given as well to permit comparison with the Registrar General's figures.* χ^2 statistical tests were used.

(a) *All Psychiatric Cases Identified During the 5½-year Period*

The term "psychiatric case" is used to include all persons who were seen by a psychiatrist as out-patients, or were admitted to mental hospital, or attempted or committed suicide. These totals do not include crimes, mental defectives outside mental hospital care, or those who remained inside a mental hospital throughout the 5½-year period.

Total psychiatric cases shown in Table I are proportionately more numerous in the Rhondda; the differences for the total cases, and for men only, are both

* For example, an admission three times during the period is counted once under "persons" and thrice under "events" in Table I. If the same person also attended an out-patient clinic, under our count of "all psychiatric cases" the total history is still recorded as one case.

TABLE I
Census Totals for the Various Counts

		Rhondda	Standardized Rhondda	Vale	P
In-patients	M	52	12.8	3	Significant <0.01
15 July, 1956	..F	39	8.9	9	Not significant
Admissions	M	87	20.9	13	Not significant
1951-56 (Persons)	F	83	18.7	33	Significant <0.01
Admissions	M	100	24.0	13	Significant <0.05
1951-56 (Events)	F	99	22.4	34	Significant <0.05
Out-patients	M	119	28.4	7	Significant <0.001
1951-56	F	150	33.6	21	Significant <0.05
Attempted suicide	M	16	3.9	1	—
1951-56	F	36	8.2	5	Not significant
Suicide	M	14	3.4	4	—
1951-56	F	7	1.6	—	—
Crimes	M	37	8.5	10	Not significant
1951-56	F	2	—	—	—
All psychiatric cases	M	190	45.5	22	Significant <0.001
1951-56	..F	224	50.6	44	Not significant

statistically significant ($p < 0.001$). The difference between the totals for women is not significant.

As the figures refer to persons irrespective of the number or type of counts under which they are recorded, an estimate of the rate of appearance of new psychiatric cases in each population was possible. Expressed as the average number of cases per annum per thousand of the population over 15 the rates are as follows; the figures are not standardized:

Rhondda: 3.81 (men, 3.67; women, 3.96, giving a male/female ratio of 0.93). Vale: 2.60 (men, 1.76; women, 3.42, giving a male/female ratio of 0.51).

These rates, considered as a measure of new psychiatric cases, will be too large as some persons would already have been in touch with psychiatric services before 1951: but the small number of duplications within the 5½-year period suggests that this may be a minor error.

(b) *Patients in Mental Hospital on 15 July, 1956*

Male patients from the Rhondda (standardized) were 4.26 times more numerous than those from the Vale ($P < 0.01$). No such statistically significant difference was shown where women were concerned, the figures being equal. These figures reflect the small proportion of men to women patients in the Vale (0.33) and the large ratio in the Rhondda (1.33). The male/female ratio for mental hospital patients in England and Wales as a whole was 0.74 in 1951 (R.G.O. 1955, Table M5, p. 134). All three men from the Vale were schizophrenics and so were 32 out of 52 men from the Rhondda. In women patients, schizophrenia was diagnosed in three out of nine cases from the Vale and in 23 out of 39 cases from the Rhondda.

(c) *Admissions to Mental Hospital (1 January, 1951 to 30 June, 1956)*

There is again a difference in the totals (persons) for men, there being 1.60 times more admissions in the Rhondda, although the difference is not statistically significant ($P=0.1$). This difference is reversed in favour of the Vale in the case of female admissions and is significant ($P<0.01$). The figure for *events* gives approximately the same results, but the difference in the case of men patients is now significant ($P<0.05$) and the significance level for women is reduced to 0.05. The ratio of male cases (events) to female cases is equal in the Rhondda (ratio 1.01) but again in the Vale the women greatly outnumber the men (ratio 0.38). The corresponding figure in 1951 for Wales is 0.75 and for England and Wales as a whole is 0.70 (R.G.O. 1955, Table M.10, p. 144).

In respect of diagnoses, rates for schizophrenic and organic and senile psychoses are similar in the two populations for both sexes. Differences are most apparent in men with affective psychoses, who occur much more frequently in the Rhondda (actual numbers Rhondda 25, Vale 1); for women the position is reversed, the Vale contributing a higher rate of cases both of affective psychoses (actual numbers, Rhondda 29, Vale 10) and of psychoneurotic and psychopathic disorders (actual numbers, Rhondda 14, Vale 7).

(d) *Psychiatric Out-patients (1 January, 1951 to 30 June, 1956)*

General practitioners in both areas referred many patients to psychiatric consultation at out-patient clinics. The totals in Table I refer to persons having at least one interview with a psychiatrist. Large and statistically significant differences were found between the totals for the two communities. Rhondda men patients outnumber those of the Vale by 4.05 times ($P<0.001$) while Rhondda women outnumber the Vale by 1.60 times ($P<0.05$). In both communities (but especially in the Vale) women patients outnumber the men, the male/female ratios being 0.87 for the Rhondda and 0.34 for the Vale. There are no comparable national figures.

When diagnoses are analysed, men from the Rhondda greatly exceed those from the Vale on all counts. The Vale yields no men with schizophrenia or affective psychosis and only five cases of psychoneurosis or psychopathy, whereas the Rhondda shows 11 schizophrenics, 16 affective disorders and 62 cases of psychoneurosis or psychopathy. In the women this discrepancy is reversed for schizophrenia (actual numbers: Rhondda 6, Vale 3); it is present but less marked for affective psychoses (actual numbers: Rhondda 31, Vale 6) but it is again very marked for cases of psychoneurosis and psychopathy (actual numbers: Rhondda 81, Vale 5).

(e) *Attempted Suicide (1 January, 1951 to 30 June, 1956)*

The figures show differences similar to those of out-patients. Men patients from the Rhondda outnumber those from the Vale by 3.87 times (a statistical test was not applied to the actual figures of 16 and 1). The difference in female cases was smaller, the Rhondda standardized total being 1.63 times the greater, which is not significant ($P<0.1$ —actual figures 36 and 5). As in the count of out-patients, women outnumber the men in both communities, the male/female ratio being 0.48 for the Rhondda and 0.21 for the Vale.

(f) *Suicide (1 January, 1951 to 30 June, 1956)*

There is a negligible difference between the suicide totals for the men. Seven cases occurred among females in the Rhondda, and none in the Vale:

the figures are too small for tests of significance. As previous studies have shown, the preponderance of females over males in cases of attempted suicide becomes reversed in cases of suicide.

(g) *Specified Crimes (1 January, 1951 to 30 June, 1956)*

Only two women (both from the Rhondda) figure in these counts. Contrary to expectation the figures for men show no statistically significant difference between the two populations.

(ii) ANALYSIS OF RHONDDA MALE CASES BY OCCUPATION

The number of male cases from the Rhondda population were large enough to warrant some further analysis. It was possible in all but 8 of these 189 cases to determine whether the individuals concerned were miners, ex-miners or non-miners.

The figures shown in Table II represent men in the Rhondda over 15 years of age who consulted psychiatric services or committed or attempted suicide during the period under review. These figures refer to persons, not events: they give a close approximation to the rate of appearance of new psychiatric cases in the populations studied.

TABLE II
Rhondda Male Psychiatric Patients by Occupation

	Psychiatric Patients	Total Population	Average Rate Per 1,000 of Population Over 15
Miners and ex-miners	86	6,513	2.37
Non-miners (including 8 never worked) ..	95	2,917	5.98
Not known	8	—	—

The group of non-miners yields proportionately more than twice as many psychiatric cases as the group of miners and ex-miners—a difference which is statistically very significant ($P < 0.001$).

When figures for the separate indices are analysed (Table III) this difference is repeated at the same level of significance for every count except those of suicides and attempted suicides. There is no significant difference in the frequency of attempted suicides in the two groups: in the case of suicide there is a reversal, the rate being over twice as high in the group of miners and ex-miners.

TABLE III
Rhondda Males by Occupation and Separate Counts

	Total Population	In-patients 1951-1956	In-patients 1956	Out-patients 1951-1956	Attempted Suicide 1951-1956	Suicide 1951-1956
Miners and ex-miners ..	6,513	31	21	56	11	12
Non-miners (including never worked) ..	2,917	53*	31	58	4	2
Not known ..	—	2	—	5	1	—

* Includes 2 who had "never worked".

However, the age distributions of the two populations at risk were very dissimilar. Among the miners and ex-miners there were progressively more in the older age groups, while in the non-miners there were progressively less. Since there is a differential risk of exhibiting a psychiatric disability in different age groups, it is possible that the differences given in Tables II and III might be related directly to this. To throw light on this point the populations at risk and the persons included in our counts were analysed by 5-year age groups, using a critical ratio test and thus holding the factor of age constant. The differences in the age range 30-54 were still found to be statistically significant ($P < 0.01$); thus at least in this age range, which included about half the cases, differences are very unlikely to be related only to the age distribution of the persons at risk in the two populations.

In the other two age ranges the differences are in the same direction but are not large enough to be statistically significant. It appears therefore that factors other than the age distributions of the populations at risk are at work.

The difference of age-distribution of these two groups *does*, however, go far to explain the disproportionately high rate of suicides among miners and ex-miners. It is known that suicide rates for men reach a peak in late middle and old age. The average age of suicides in the mining group is 57; the ages of the two instances in non-miners are 33 and 34. The average ages of the populations at risk are 47.8 and 34.9 years respectively, with a loading of the older age-groups in favour of the miners.

An analysis of the diagnoses of men admitted to mental hospital over the 5½-year period shows that the main differences between the two groups are in the categories schizophrenia and psychoneuroses; the rate of occurrence of schizophrenia being four times greater and that of psychoneuroses nine times greater among the non-miners (actual figures: "mining" 7 schizophrenia, 4 psychoneuroses; "non-mining" 12 schizophrenia, 18 psychoneuroses). The "depressive" and "organic" groups show only small differences. Because of the small numbers and different age distribution of the populations at risk these figures can only be suggestive.

DISCUSSION

The salient points emerging from this survey can be briefly summarized as follows: psychiatric cases were found to occur more frequently in the Rhondda than in the Vale, but whereas in the former area the sex ratio approaches equality, in the Vale cases were twice as numerous in women as in men. As compared with national figures, Rhondda men were over-represented and Vale men were under-represented among patients admitted to, or remaining in, mental hospitals.

Men from the Rhondda sought psychiatric out-patient advice four times more frequently than did men from the Vale. In both areas women out-patients outnumbered the men; this was particularly evident in the Vale. Cases of attempted suicide showed a similar distribution, but actual suicides occurred with equal frequency among men of either community. Men from the two areas were involved with equal frequency in crimes of violence.

On sub-dividing the Rhondda men into "miners and ex-miners" and "non-miners" it was found that psychiatric cases (in all counts except that for suicides) occurred more frequently in the minority group of "non-miners". This preponderance could not entirely be accounted for by the differing age-range of the two groups; but the relatively high rate of suicides in the former

group could be so explained. In terms of diagnoses, the "non-miners" yielded more than their share of cases of schizophrenia and psychoneurosis, but equal numbers of affective disorders and organic syndromes.

Some of these findings conform to expectation. It has repeatedly been shown that admissions to mental hospitals occur more frequently from urban than from rural areas. For example, standardized rates for first admissions in New York State (Malzberg, 1940) have shown urban/rural ratios of 1.8 to 1 for men patients and 1.5 to 1 for women. Figures for admissions to mental hospitals in England and Wales in 1951 from Country Boroughs and Rural Districts show less marked differences: the ratios for men are 1.48 to 1 and for women 1.32 to 1 respectively (R.G.O. 1955, Tables M.21(a) and (b)).

In this survey the ratio of male admissions to hospital from the Rhondda to those from the Vale is 1.60 to 1, but the rate of admission of women patients is actually higher in the Vale than in the Rhondda.

Male patients from the Rhondda have accumulated in mental hospital so that they are four times more numerous than the corresponding figure from the Vale; but in spite of a much higher admission rate the women patients from the Vale have *not* accumulated in hospital to a corresponding degree. Any consideration of totals of patients remaining in hospital must be qualified by the reminder that some of these patients were admitted many years ago, from populations which may have differed in some respects from those of the present study.

In analysing rural-urban differences in hospital rates in Ontario, the authors of a careful study (Buck, Wanklin, and Hobbs, 1955) compared the severity of patients' symptoms on admission, and reached the conclusion that "the excess urban admission rate arises almost entirely from a greater urban tendency to hospitalize cases whose symptoms could be tolerated in the community". This conclusion, if it is relevant to the present findings, would suggest that the Rhondda population is more intolerant of incipient psychiatric symptoms in its men, and the Vale in its women-folk. The Vale appears readier to receive ex-hospital patients back into the community.

There is a conspicuous difference in the frequency with which men from the two communities have consulted psychiatric out-patient clinics: the Rhondda men preponderate greatly in all diagnoses. This preponderance is much greater than that for admissions to mental hospital (which can be regarded as indicating a more serious complaint than mere attendance as an out-patient) and it disappears altogether when actual cases of suicide are counted. It appears therefore that men from the Rhondda have been readier to present with the minor forms of mental disorder than have men from the Vale. The same is true of women from the two areas; women out-patients from the Rhondda were 1.6 times more numerous than those from the Vale and this preponderance was not due to cases of schizophrenia or affective disorder, but primarily to an increase in the psychoneuroses.

It appears therefore that recourse to psychiatric advice occurs more frequently in the Rhondda; and that in the Vale women seek this recourse much more readily than do the men. This may indicate that women are more dispensable in the rural community. In view of the frequency with which the coal-mining community becomes the target of unfavourable comment, it is interesting to note that it was the *non-miners* in the Rhondda who contributed more than their share to every index of psychiatric disorder except actual suicides. The relatively high rates among non-miners (which are only partly accounted for by their lower age-range) could be regarded as an instance of

the vulnerability of the minority group in a community (Faris and Dunham, 1939); but a more likely explanation is that of adverse selection to a less favourable occupation—an explanation which has been adduced to account for high rates of psychiatric breakdown among seamen in Norway (Odegaard, 1956; Sundby, 1956). Mining is a strenuous, dangerous, but relatively well-rewarded occupation. In the Rhondda miners are the aristocrats among manual workers, so that one can expect a higher incidence of physical and emotional handicap among those who elect to take lighter and less well-paid jobs instead.

Speculation is easy, but a linking together of social pressures and psychiatric disabilities in causal terms must await fuller analyses (*i*) of the social structure and value systems of the communities (with particular reference to prevailing attitudes to mental illness), (*ii*) of the circumstances leading to the emergence of the declared cases, and to the "carrying" of undeclared illnesses in each community, and (*iii*) of the clinical features of the illnesses themselves. Towards such a study the present survey can serve only as a pointer.

SUMMARY

1. A count was made of psychiatric cases, attempted suicides, suicides and crimes of violence occurring during a 5½ year period in two defined communities in South Wales, a mining valley and an agricultural area.
2. Cases occurred at the rate 3·8 per 1,000 of population over 15 per annum in the mining valley and 2·6 per 1,000 per annum in the agricultural area.
3. In the mining valley, admissions to mental hospital were as frequent among men as among women; suicides were twice as frequent among men. Women outnumbered men in out-patient attendances and attempted suicides.
4. In the agricultural area, women greatly outnumbered men in all psychiatric counts except suicide.
5. Crimes of violence occurred with equal frequency among men of either community.
6. On analysing the Rhondda male cases by occupation, it was found that the larger group of miners and ex-miners showed a significantly higher rate of suicide than did non-miners: this could be accounted for by their higher mean age.
7. The group of non-miners contributed a disproportionately high number to all other indices of psychiatric disorder.
8. The relation of these findings to sociological differences between the communities is discussed.

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THE PAVLOVIAN THEORY OF HYPNOSIS: AN EVALUATION*

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EXPOSITION

PAVLOV's theory of hypnosis is, perhaps, the first theory of hypnosis to be essentially derived from physiological experiments. Arising out of his experiments with dogs, the theory hopes to explain the hypnotic phenomena in human beings.

While engaged in experiments on delayed conditioning, Pavlov noticed that some dogs develop a state of drowsiness after the onset of the CS and lasting until the unconditioned stimulus is given; such states of drowsiness were also observed in animals who had been used for conditioning experiments which started some time after the animal was fastened to its stand. By the repetition of an already extinguished stimulus or the repetition of a negative stimulus established during differential conditioning, drowsiness could also be aroused. Repetition of an indifferent stimulus in a continuous monotonous manner was also observed to produce the same reaction. A sufficiently prolonged continuation of any of the above operations led to deep sleep.

Pavlov called this a state of inhibition between wakefulness and sleep. He called this a state of inhibition for reasons which shall be clear later. The most obvious reason, however, was that it could be produced by the repetition of a stimulus which has acquired inhibitory property.

The following quotation from Pavlov indicates the relation between sleep and inhibition:

"Internal inhibition† and sleep are one and the same process . . . *inhibition* is a partial, fragmentary, strictly localized sleep confined within definite boundaries under the influence of the opposing process: that of excitation; sleep, on the contrary, is an inhibition which has spread over a great section of the cerebrum, over the entire hemisphere and even into the underlying midbrain." (Pavlov, 1928.)

Thus, sleep and internal inhibition are similar, but not identical.

The phases between wakefulness and sleep are called hypnotic phases by Pavlov. There can be obtained gradual intermediate states of inhibition (hypnosis) ranged from those hardly differing from wakefulness to states tending to deep sleep. These states differ in their intensity and extensity of inhibition in the cerebral cortex (Ivanov-Smolensky, 1952, p. 112). As the application of the inhibitory stimulus continues, inhibition begins to spread; unless otherwise directed by verbal suggestions of the hypnotist, gradually the motor areas of the cortex come under inhibition and catalepsy and paralysis

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† For those less familiar with Pavlov's system, internal inhibition is an active, elaborated conditioned inhibition: extinction is an example. In contrast, external inhibition is a passive unconditioned inhibition. A very loud tone abolishes the conditioned reflex: this can be explained by external inhibition. Both have the same neurological basis.

may be observed. When inhibition also spreads to the sensory areas and in fact covers the whole cortex, normal sleep develops.

Pavlov noted interesting changes as the state of inhibition deepened. In the first stage of hypnosis (in the animal) both the weak and the strong stimuli act exactly alike in eliciting the CR (Conditioned Response). This is called a state of equalization. Then comes a stage where the weak stimulus is effective, but the strong stimulus is either not at all effective or has a very weak effect. This state is aptly described as the paradoxical phase. In the third stage, called the ultra paradoxical phase, only the previously elaborated inhibitory agents have a positive effect. Finally, a state of complete inhibition characterizing sleep develops. Pavlov seems to imply that the above-mentioned states develop in the same sequence always. Ivanov-Smolensky mentions experiments where this order is not always maintained.

Inhibition, in the Pavlovian system, is a protective mechanism necessary for the survival of the organism. Excitation and inhibition are at opposite poles. When a nerve cell is stimulated beyond its physiological capacity, inhibition sets in. So also, when an extinguished CS (Conditioned Stimulus) is applied continuously, inhibition develops, protecting the cortical cells from "unnecessary" excitation. A partially decorticate animal develops a state of inhibition more quickly than a normal one, because its remaining cortical cells are liable to be more easily exhausted. Thus, the organism is protected from any stimulations that are not relevant for its survival. Sleep, the manifestation of complete cortical inhibition, is, therefore, a valuable protective mechanism. But how does Pavlov explain the state of hyper-suggestibility in hypnosis? The fact is: under hypnosis, there is a selective inhibition of certain parts of the cortex with points of excitation maintained by the suggestions of the hypnotist. It can best be described by quoting Pavlov: "What is suggestion and auto-suggestion? They are concentrated excitation of a definite stimulation, sensation or its trace, a representation, now in evoked emotion, i.e. excited from an internal connection, an association, excitation having been given a predominant, illegitimate and irrepressible significance . . . In hypnosis, we have a diminished positive tonus (in the cortex) as a consequence of irradiated inhibition. When on such a cortex a word or order of the hypnotizer is directed to a definite point the stimulus concentrates the excitatory process in the corresponding point and is immediately accompanied by negative induction, which causes it, without the slight opposition, to spread over the whole cortex; this is why the word or the order is completely isolated from all influences and is made an absolute, irrepressible, fatally-acting stimulus, even after the subject has returned to the waking state" (Pavlov, 1941, pp. 108-109).

CRITIQUE

Pavlov is not the only one who holds that hypnosis and sleep have some essentially common elements. Many past and contemporary investigators implied the existence of such a relationship.

Bechterew (1906) regarded hypnosis as a modification of normal sleep induced by suggestions under appropriate physiological conditions: "Hypnosis is nothing, but a modification of sleep." Schilder and Kauders (1927) also maintained in their monograph that hypnosis is a suggested sleep. They were confident that many of the phenomena in hypnosis could be explained by the psychology of sleep. Brown (1935) was of the same view, apparently resembling Pavlov's. Koster (1954) considered hypnosis as a special kind of sleep. None

of the above investigators *equate* sleep with hypnosis. Barber (1956), however, has reported an experiment where he gave seven suggestions (usual suggestions acceptable in a light to medium depth of hypnosis) to subjects under light sleep and hypnosis. "... There were no significant differences between the subject's responses on the seven tests of suggestibility when the subjects were 'lightly sleeping' and after they had been subjected to hypnotic induction." If the degree of suggestibility is taken as an index of hypnotizability (as it is implied in most of the scales to measure hypnotizability), it would appear that light sleep and hypnosis, within the limitations of the reported study, are not significantly different.

Most Russian studies are biased to the Pavlovian standpoint on hypnosis. Assuming that hypnosis is a state of inhibition, Strelchuk (1953) shows how the hysterics, who have a weak second signalling system, can be more easily hypnotized by verbal suggestions; while the psychasthenics, with a weak first signalling system, are more susceptible to hypnosis through rhythmic, photic or dermal (especially thermal) stimuli. Evidently he does not question Pavlov's view on hypnosis. Korotkin and Suslova (1955) similarly mention that verbal suggestions affect subjects differently, the difference being most noticeable in post-hypnotic suggestion. The type of signalling system, they think, may explain some of these cases.

A field of controversy has been the possibility of establishing or "recalling" CRs during hypnosis. If hypnosis is a state of inhibition, it is argued, previously established CRs should disappear or new CRs should be difficult to form during hypnosis. Arguing, apparently, in this strain, Scott (1930) concluded that hypnosis is not a state of inhibition resembling sleep, because CRs were formed more easily under hypnosis in his experiment. But he did not make sure that the subject was in a really deep state of hypnosis most approximate to sleep, so far as inhibition was concerned. Most of the studies which show a facilitation of conditioning under hypnosis are liable to the following criticism: first, the techniques of many of these studies are faulty; the depth of hypnosis has not always been ascertained or mentioned. Where a mention has been made of the depth of hypnosis, adequate checks do not seem to have been made as to whether the same depth has been maintained throughout the experiment or in every session of the experiment. Only a very deep state of hypnosis can be expected to decrease conditionability, because after all, inhibition is partial in hypnosis. Secondly, it may be argued that with a lesser depth of hypnosis, it is only natural that conditioning would be facilitated. It is well known that attitudes interfere with human conditioning (Razran, 1936, 1939). Under hypnosis, when the subject is less critical of what is happening, conditioning may be hoped to be facilitated. Why a really deep state of inhibition is needed to hamper conditioning is shown by two drug experiments.

Sterling and Miller (1941) tried to condition cats under four progressive stages of anaesthesia. Although a small number of cats showed decreased conditioning in the first level (23 per cent.), only on the fourth level was conditioning not possible for any of the cats. Thus it really required a deep level of anaesthesia (and so, inhibition) to abolish conditioning. Headlee and Kellog (1941) found that conditioning proceeded at a lower level, but by no means disappeared under doses of nembutal.

There are quite a few Russian studies showing that conditioning or some other functions suffer under hypnosis. All of them used somnambulistic hypnosis. Maïorov (1950) has found that sensory chronaxie is lengthened during somnambulistic hypnosis; inability to do simple additions, anaesthesia

and absence of blinking were also observed. He concludes that these are due to an irradiation of inhibition and dissociation—both characteristic of hypnosis. Korotkin and Suslova (1951) report that in the somnambulistic phase, CRs sometimes cannot be established; differentiation is equally impossible; and if CRs can be established at all, they are slowly acquired and have a longer latency. The same authors in another paper (1953) report the disappearance of previously established CRs under somnambulistic hypnosis. In a stage of lesser trance depth, such CRs do appear. Korotkin and Suslova also found that by giving a hypnotic suggestion to inhibit UCS (Unconditioned Stimulus), the CS was inhibited along with the UCS. They explain this in terms of irradiation of inhibition (1955c). The same authors have shown, as did Maiorov, how simple counting and additions become full of errors when worked out under hypnosis. The subjects were given equivalent addition and counting tasks in the state of wakefulness and during somnambulistic hypnosis. They performed definitely worse in hypnosis.* The authors conclude from this that under somnambulistic hypnosis higher nervous activities are affected by inhibition. The carrying out of post hypnotic suggestions is also explained by them in terms of inhibition (1955a, b). All the experiments mentioned above are consistent with Pavlov's view.

The EEG has been used in several studies to settle the issue whether hypnosis is a state of wakefulness or of sleep. Before we consider some of them, let us point out the basic fallacy that may vitiate such attempts to settle the question. The method of many EEG studies has been to obtain records from the same subject, during sleep, hypnosis and wakefulness. When they find that the brain waves under hypnosis resemble those of wakefulness more than they do those of sleep, the usual conclusion has been that hypnosis is not sleep. This neither supports nor opposes the Pavlovian concept of hypnotic phases as intermediate states of inhibition between wakefulness and sleep. It has never been claimed by Pavlov or Pavlovians that hypnosis *is* sleep. In fact, in a great many ways, they are considered different. Even under the deepest possible level of hypnosis, a contact is maintained between the subject and the hypnotist. There are still some cortical points which are "awake". In this sense, the cortex is only partially inhibited. In sleep, even under light sleep tending to a deep one, the whole cortex is under a state of inhibition. The inhibition is perhaps only less intense during light sleep. Attempt at communication hampers development of sleep, while possibility of communication is an essential characteristic of hypnosis. So, if sleep may be visualized to vary in a dimension of intensity of complete cortical inhibition, hypnosis cannot be described in such a dimension. The intensity of inhibition in those cortical areas inhibited under hypnosis may be as great as that found in deep sleep. But still there would be certain cortical points outside that area: some of those points may be under acute excitation by the verbal suggestion of the operator. Seen from this angle, hypnosis and sleep appear to lie in orthogonal dimensions.

We do not know, then, whether the partial wakefulness of the cortex during hypnosis creates a special problem in comparing the EEG records obtained in sleep and hypnosis with a view towards settling the original issue posed earlier.

* There are many studies on learning and memory during hypnosis which report either no deterioration or an improvement of these functions. Weitzenhoffer (1953) has written a good review of them, so we need not review them here. Because even such well-written books, like Hull's (1933) or Weitzenhoffer's, do not contain any reference to Russian or other studies supporting Pavlov's view, we thought it appropriate to bring these evidences to light. This has been adopted as almost a general policy throughout the present paper.

With this orientation, let us consider some relevant EEG studies. Just before going into the literature on hypnosis and EEG we may mention here an experiment showing how Pavlov's concept of cortical inhibition is demonstrable electroencephalographically. Morrell and Ross (1953) and Morrell (1956) could show that whenever any of the three types of inhibitions (manifested during extinction, delayed and differential conditioning) were induced, a delay in cortical conduction was occurring. They used conditioning of cortical alpha rhythm for their CR.

Darrow, Henry, Brenman, and Converse (1950), in two studies, find a significant difference between states of wakefulness and hypnosis by using EEG. "Analysis of available double speed (monopolar) EEG records obtained on 11 fair to good hypnotic subjects shows, with only one exception, an increase of average in phase peak to peak correspondence between waves in occipital and motor areas." The increment for the hypnotic state was significant below 1 per cent. level. The "records from some of the same subjects while going to sleep show a similar increase in frontal-motor parallelism during drowsiness or early phases of sleep". Hypnosis was, thus, significantly different from wakefulness and only resembled sleep in the early stages. Lundholm and Löwenbach (1942-43) studied the changes in alpha activity under hypnotic suggestions of blindness and deafness. They found that alpha activity was present only when there was an actual absence of stimulation. When blindness was suggested and a light held in front of the subject's eyes, the alpha rhythm disappeared; they reappeared only when the light was actually removed. This shows that physiological modifications, at least in the cortical level, if any, are not made by hypnotic suggestions. The same conclusions could be derived from a study of hypnotic anaesthesia and analgesia (Brown and Vogel, 1938). If a light is held before the closed eyes of the sleeper, perhaps the alpha waves would not disappear, if they were present. But under suggested hypnotic blindness, in the same conditions, they did disappear. From this it would be erroneous to conclude that hypnosis is not a state of inhibition resembling sleep. For during sleep, if it were possible to keep the eyes open without awakening the subject as is done under hypnosis, it is probable that holding a light before the open eyes of the sleeper would block the alpha activity. Furthermore, under hypnosis, suggestion may be able to inhibit only the *perception* (interpretation of sensation) rather than the physiological outcomes of sensation. A dissociation between sensory and association areas may be the most likely neurological explanation.

Barker and Burgwin (1948, 1949) made a new approach to the problem by distinguishing hypnosis from sleep and hypnotic sleep. They found that when trance was induced without any mention of "sleep", "relax", etc., the brain wave patterns did not resemble sleep at all. But when the subject was asked to relax, his attention narrowed down and suggestions of sleep were given. "Sleep indistinguishable electroencephalographically from normal sleep in various stages" could be produced and terminated. "We have repeatedly observed that suggestions which minimize sensory stimuli and ensure maximal muscle relaxation are associated with changes in the brain wave patterns towards that of sleep." It may be remarked that most induction methods use relaxation and suggestions of narrowing down of attention. So it can only be asked whether the brain wave patterns characteristic of sleep do not disappear when communication is attempted with the subject. Sleep, according to these authors, "is characterized by the absence of active relationship with the environment". If the operator's suggestions can be accepted as a part of the environment,

hypnosis cannot, perhaps, be described in identical terms. Dynes (1947) seems to have overlooked this. He obtained EEG records continuously from subjects during the induction of hypnosis and perhaps implicitly argued that as the subject passes from wakefulness to hypnosis the EEG records would progressively resemble those of sleep. This was, obviously, not found; he does not even mention whether the subject was taken to a somnambulistic stage.

Summarizing the researches in this field, Chartok and Kramraz (1955) arrive at the conclusion that EEG studies are not yet in agreement about electroencephalographic similarity between sleep and hypnosis. They also report an experiment done by them on good hypnotic subjects. Again, only in some subjects did brain waves resembling those during sleep appear. An absence of operator- and subject-contact could allow the sleep pattern to be retained in some.

In concluding this section, we only mean to reiterate our standpoint outlined at the beginning of the section: we do not know whether the partial wakefulness of the cortex during hypnosis may account for all the difference between EEG records obtained under sleep, wakefulness and hypnosis.

AN EXPERIMENTAL SUPPORT

It has been mentioned before that Pavlov's dogs could develop states of drowsiness under monotonous stimulation. On subsequent occasions, the dogs became drowsy in an increasingly shorter time. Thus, at last, a dog may develop drowsiness as soon as it is brought into the laboratory or fastened to its stand. Two distinct points could be noticed: (i) a state of drowsiness (as a result of inhibition) can be produced by laboratory techniques; (ii) with subsequent practice, the development of such a state may appear as a conditioned response to the environment of the laboratory itself.

These two findings are from studies on dogs. Can these be obtained with human subjects? What, if any, is the relation between the speed of development of such a state and the depth of hypnosis attainable for a certain subject? The experiment to be reported was designed to answer these questions.

During an experiment on hypnosis and conditioning, it was discovered that some subjects could not be made to wake up easily after hypnosis and some of them became so drowsy during an experiment on eyelid conditioning that they had to be reminded frequently to keep their eyes open. One subject showed this trend so strikingly that it could not be overlooked. He was hypnotized, but could not be awakened in the usual manner (counting slowly up to 15 and asking the subject to wake up at the end of the counting). Commands, persuasions, mild shaking, could not wake him. After some shouting and severe shaking, the subject finally woke up slowly. E (experimenter) assumed that the subject had fallen asleep; but upon asking the subject, he said that he was listening to all the persuasions of E to awaken him, but just could not pull himself out of that state. During the conditioning experiment, he began to feel so extremely drowsy that he could not keep his eyes open, and the experiment had to be discontinued. Later, he reported that in spite of his best attempts, he could not keep his eyes open. This case, an extreme of similar cases, led to the experiment to be described below.

METHOD

Four subjects, whom we shall call OF, RA, ST and MA, were used. RA was the case described above. OF and RA had high scores in hypnosis: RA had the highest possible score, 6; OF had a score of 5; ST and MA scored

low in hypnosis: 1 and 2 respectively. The lowest possible score was 0 and the highest was 6.

The following device was used to produce inhibition. In a sound-proof room, usually used as a conditioning laboratory, S was asked to sit comfortably in a dentist type chair. He had, in front of him, a booth with a little red light (.5 watt) in the centre. He was instructed to keep looking at the red light all through the experiment. A continuous pure tone of 1,110 cs. per second, an intensity of 10 decibels above the subject's auditory threshold, was transmitted to the pair of earphones which the subject was wearing. The tone was regularly discontinued for .5 second in every 30 seconds. S had been asked to blink when he perceived this break in the tone. S was wearing a pair of spectacles with plain lenses; on the lens for the right eye, a tiny photoelectric cell was mounted and was connected to an appropriate apparatus, and then to a tracing pen. Besides this pen, there was another pen acting as an event marker. Both the pens were parts of a recording milliammeter. Thus every time a break in the tone was made, the event marker traced an elevated curve for .5 seconds and if the subject blinked, the other pen traced a rather sharp rise. E had a switch which, when pressed, would discontinue the tone for .5 seconds, after which the tone would resume automatically. E with all the apparatus was in one part of the room, separated from S by a one-way screen.

S was not told about the purpose of the experiment. He was told that he is expected to sit relaxed and to blink to the break in the tone.

Since the experiment was of an exploratory nature, a rigid common procedure could not be fixed from the beginning. It was only broadly decided that the experiment should be carried on for 20 minutes and if an S did not miss a single chance to blink to the break in the tone during that period, the session should come to an end. If S began to show signs of drowsiness and failed to blink, the session was to continue until he did not blink to 10 successive breaks. He was to be taken out of the laboratory, given ten minutes of performance on the pursuit rotor and again brought back for the next session. In this way, the experiment was to continue until S did not blink to the first break in the tone.

RESULTS

A summary of the results are to be found in the accompanying table. It will be seen that OF could not blink 30 seconds after the 4th session started and RA fell into a state of drowsiness as soon as the 2nd session began. In fact, he later reported that he felt sleepy as soon as he entered the laboratory for the 3rd session. Both of these subjects were seen sitting in the chair, with their heads drooping and eyes closed, after they had stopped blinking. RA was snoring

TABLE

Subjects		Hypnotism Score	Trials* and Minutes after which Blinking Stopped				
ST	..	1	Did not stop.				
MA	..	2	Did not stop.				
			Sessions				
			i	ii	iii	iv	
OF	..	5	12	10	3	1	(trials)
			6	5	1.5	.5	(minutes)
RA	..	6	12	0	0	—	(trials)
			6	0	0	—	(minutes)

* Every time a break in the tone occurs is called a trial; each trial occurring once in every .5 minute (30 seconds).

audibly in the third session. In contrast, ST reported having felt only bored. MA felt sleepy, but said that he did not think that he would have really fallen asleep had the experiment continued longer.

DISCUSSION

The results need little discussion. Our first objective was to show that a state of drowsiness, analogous to the one observed in Pavlov's dogs, could be produced in human subjects. Perhaps we can say that we have been able to demonstrate it unequivocally. We have further shown that in those in whom this state of drowsiness developed, it began to take a shorter time with practice, so much so that the mere sight of the laboratory environment initiated it—our subject RA developed drowsiness in less than 30 seconds. Pavlov had also noticed similar phenomena in his dogs. To support hypnosis as a state of inhibition, we find that the two subjects who did not develop such a state scored low in hypnosis—indeed, they are to be classed as un hypnotizable if we were to classify dichotomously. (A person scores two if he does not accept any of the five hypnotic suggestions, apart from the post-hypnotic suggestions, but feels relaxed, heavy and slightly drowsy; a score of one is given when he feels just relaxed.) A correspondence can also be noted between the hypnotic scores and the reaction to the experiments:

ST Hypnosis score: 1, felt bored.

MA Hypnosis score: 2, felt sleepy.

OF Hypnosis score: 5, found sleeping after 30 seconds.

RA Hypnosis score: 6, found snoring.

The number of cases is too small to apply any statistical technique to demonstrate a relationship. It would, however, be permissible to expect a positive correlation between depth of hypnosis attained and the speed of the development of this state of drowsiness under monotonous stimulation. We also found indications of a positive relation between the depth of hypnosis and the speed of conditioning to the monotonous environment of the laboratory which produces the state of drowsiness. Does it imply that a correlation between hypnotizability and conditionability exists? We consider it a likely hypothesis.

We have been calling the experimentally produced state under which the subject could not keep his eyes open to be able to blink a state of drowsiness. This is clearly a superficial nomenclature. If we follow the Pavlovian terminology, it can best be described as a state of experimentally produced inhibition intermediate between wakefulness and sleep. This is a learned state inasmuch as it is amenable to improvement with practice. If a correlation is found between this and hypnosis, and is interpreted as meaning a basic similarity between the two, hypnosis may be defined as a learned state of inhibition and hypnotizability an ability to learn to develop inhibition.

SUMMARY

A brief exposition of Pavlov's conception of hypnosis as a state of inhibition intermediate between wakefulness and sleep was made. Evidence mainly supporting this view was presented. Some relevant EEG studies were examined. Some of them appeared to have disproved the identity between sleep and hypnosis. This was not regarded as evidence against the Pavlovian view of hypnosis. A small experiment carried out by the author was described. It demonstrated the possibility of the development of a state of inhibition (drowsiness) in human subjects by monotonous application of sound and light stimuli in a generally monotonous laboratory condition. Pavlov had demonstrated similar phenomena on dogs. The results of the experiment also suggested that such a development of inhibition improves with practice and may be correlated positively with an increasing degree of hypnotizability.

On the whole, it is felt that there is enough evidence to make out a case for supporting Pavlov's view of hypnosis as a state of partial or selective inhibition.

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CHEMICAL AND PATHOLOGICAL FINDINGS IN A CASE OF LATE INFANTILE AMAUROTIC FAMILY IDIOCY OF THE BATTEN TYPE

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THE customary classification of the amaurotic family idiocies according to the time of life at which neurological signs first appear has obvious limitations when consideration is given to cases occupying an intermediate position between the well-established infantile form of 'Tay-Sachs' and the juvenile form first described by Batten (1903). It was to this group of atypical cases that Bielschowsky (1913-14, 1920) gave the name "late infantile". This term is convenient clinically but when more examples of the variant had been reported it became evident that the group of cases so designated was not homogeneous. Wyburn-Mason (1943) was able to show in a large clinical material that many of these intermediate forms belonged either to families affected by Tay-Sachs' or by Batten's disease and that in an individual case the ophthalmological findings were usually of decisive importance in classification. The existence of a precocious variant of the classical "juvenile" type of amaurotic idiocy was thus clearly established. Klenk's (1939) discovery that the nerve cells in Tay-Sachs' disease contain large amounts of a glycolipid subsequently called "ganglioside" has provided a further valuable criterion in distinguishing this form of lipidosis from other members of the group. The present case, an example of the precocious juvenile type of amaurotic idiocy or, as we prefer to name it, the sub-acute form of Batten's disease, is presented as a further contribution to this subject.

CASE REPORT

Clinical History

J.V., a female, died at the age of 4 years and 8 months. She was the second child, the elder and the two younger siblings being normal. The parents were of Gypsy stock, healthy and not consanguineous. The pregnancy and delivery were unremarkable and the birth weight was 6 lb. By the age of 2 years it was evident that a serious arrest of development had occurred. She was incapable of sitting up or doing anything for herself and speech was limited to "Mum" and "Dad". When seen as an out-patient she was considered to be a case of primary oligophrenia with microcephaly. She was not thought to be blind and no ophthalmoscopic examination was made. The child's condition remained more or less stationary and at no time did she have any fits. On the day of her death she vomited continuously and died suddenly.

Post-mortem Findings (22 hours after death):

A small, severely wasted female child, weighing 5,550 g. Subcutaneous fat completely absent, only a trace of fat present in mesentery. Severe dental caries. All organs small for the child's age: heart 45 g., right lung 80 g., left lung 60 g., liver 240 g., spleen 20 g., adrenals

10 g., kidneys 40 g. each. Stomach contents are present in the air passages. Except for patchy consolidation in the lungs the viscera show no abnormality.

The skull appears small for the child's age, but is not abnormal in shape. The dura is normal. The brain does not fill the cranial cavity and there is an excess of cerebrospinal fluid over the surface. The leptomeninges are normal.

The brain (284 g. after fixation) is small, firm and rubbery. The convolutions of the cerebral cortex are narrow and are separated from each other by widely gaping sulci (Fig. 1). On coronal sections the cortex appears thin and poorly demarcated from the underlying white matter. The ventricles are moderately dilated. The cerebellum is small and the thin, sclerotic folia are separated by wide fissures (Fig. 2). The brain stem shows no abnormality.

Histology

Lung: Acute confluent bronchopneumonia. A few cells, both in the alveolar walls and in the lumina, contain granular P.A.S. positive material.

Liver: The general lobular architecture and the reticulin pattern are normal. In the portal tracts there are small aggregations of P.A.S. positive storage cells. Similar isolated cells can be seen in the sinusoids, their nuclei resembling those of Kupffer cells. The liver cells appear healthy and do not contain any visible deposit.

Spleen: The general architecture is well preserved, but the lymphoid follicles are small and devoid of germinal centres. The pulp shows moderate reactive hyperplasia. The sinusoids appear normal. The reticulin pattern is normal. There is no congestion or fibrosis. Numerous aggregations of storage cells can be seen both around the larger vessels in the trabeculae and around the arterioles in the follicles (Fig. 3). There are also minor aggregations in the pulp. The sinus endothelium contains a few flecks of P.A.S. positive material.

Lymph node: The general architecture is somewhat blurred, the follicles are poorly outlined and devoid of germinal centres, but the reticulin pattern is normal. There are a few small aggregates of storage cells scattered throughout the node and not related to any particular structure.

Thymus: Consists predominantly of lymphoid tissue, among which numerous clumps of storage cells are scattered, mostly near the periphery of the lobules in the neighbourhood of the fibrous trabeculae.

Pituitary: The anterior lobe is normal. In the posterior lobe flecks of lipid can be seen in the processes of the nerve cells.

Adrenal: The cortex is normal. The medulla contains a few isolated P.A.S. positive cells.

Pancreas: Perivascular aggregations of storage cells can be seen in the fibrous septa. The acini and islets are normal.

Kidney, heart and skin: Normal appearances.

Brain: Representative frozen sections of the cerebral cortex, basal ganglia, brain stem and cerebellum were stained by standard methods for nerve cells, axis cylinders, myelin, fibrillary neuroglia and lipid. A number of special histochemical procedures were also carried out (Table I).

Cerebral cortex: There is a generalized and severe loss of nerve cells associated with a marked disturbance of the lamination of the cortex and a reduction in its depth. The great majority of the remaining cells, exclusive of the neuroglia, are rounded structures with a basophilic, granular cytoplasm and one, or sometimes two, small pyknotic nuclei. These cells are sometimes irregularly oval or triangular in shape and measure 10–20 μ in diameter, the smaller forms predominating. Their outlines are crenated owing to the coarse granularity of their contents and they possess no neurofibrils and usually no processes (Fig. 4). It is, however, possible to trace transitional forms between these macrophage-like structures and some of the larger cells which are more obviously degenerate neurones displaying shrinkage rather than swelling. Here and there in the cortex are nerve cells showing the familiar type of swelling found in amaurotic idiocy, the cytoplasm being pear-shaped, the granular contents finely dispersed and the peripherally displaced nucleus being better preserved than in the pyknotic cells just described. The largest numbers of such cells occur in the precentral gyrus where the giant cells of Betz show conspicuous distension. The cortex is also rather better preserved in the hippocampus than in other areas which have lost their individual features through cell loss and the gross distortion in shape of the surviving neurones. Throughout the whole atrophied cortex the numbers of astrocytes are increased and there is a dense fibrillary gliosis. Some of the microgliaocytes show small flecks of granular material in their processes but no compound granular cells containing neutral fat are present in the grey matter.

The *subcortical white matter* is also considerably shrunken and shows only a sparse network of myelinated fibres most of which are irregularly swollen and beaded (Fig. 5). A well-marked cellular and fibrillary gliosis is present (Fig. 6). Evidence of recent myelin breakdown was found only at the occipital pole where numerous fat granule cells can be seen.

Basal ganglia: The thalamus shows severe cell loss in the medial and ventrolateral nuclei, the anterior nucleus and the centrum medianum being relatively well preserved. The affected parts show dense gliosis. The surviving cells all show lipid storage as also do those of the striatum and pallidum where there is no apparent cell loss. Other nuclear masses in the sub-thalamic and hypothalamic regions show similar cell changes. Myelination of the striatum and pallidum is fairly good while that of the internal capsule is poor. The optic tract is fairly well

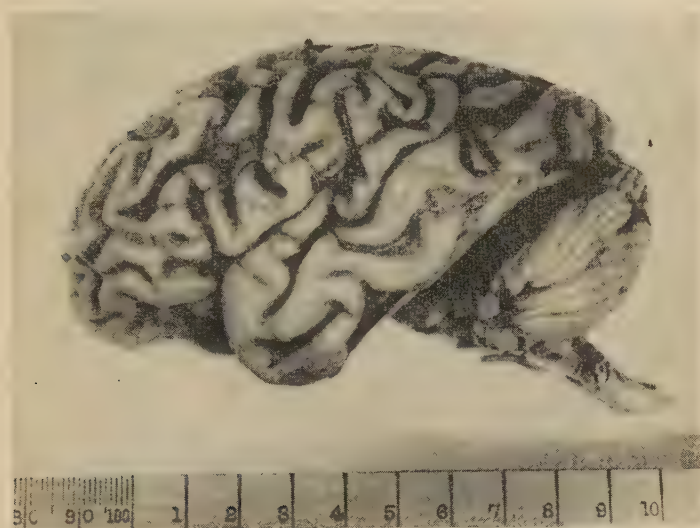


FIG. 1.—Lateral view of brain: atrophy of left cerebral hemisphere.

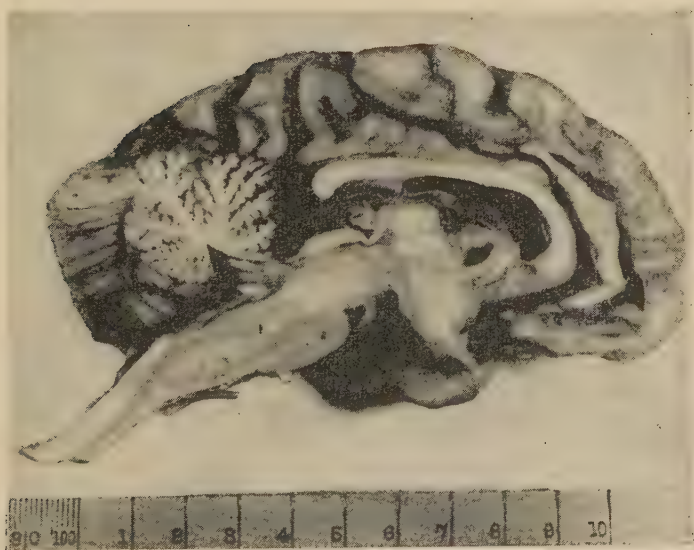


FIG. 2.—Sagittal section of brain: cerebral and cerebellar atrophy and dilatation of ventricular system.

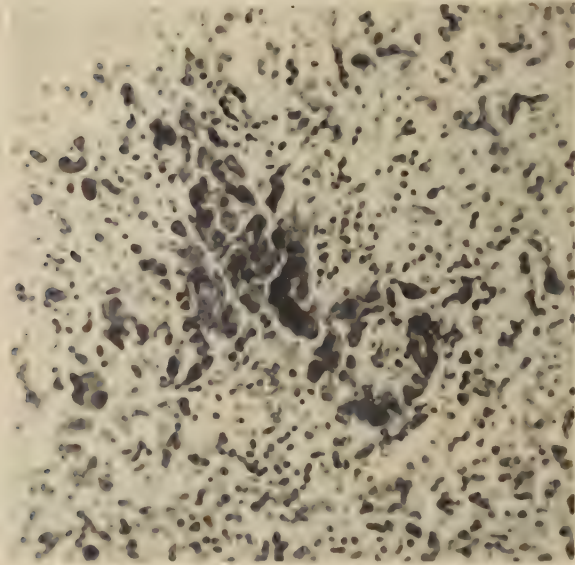


FIG. 3.—Spleen: perivascular aggregation of histiocytes laden with lipid. P.A.S. $\times 240$.

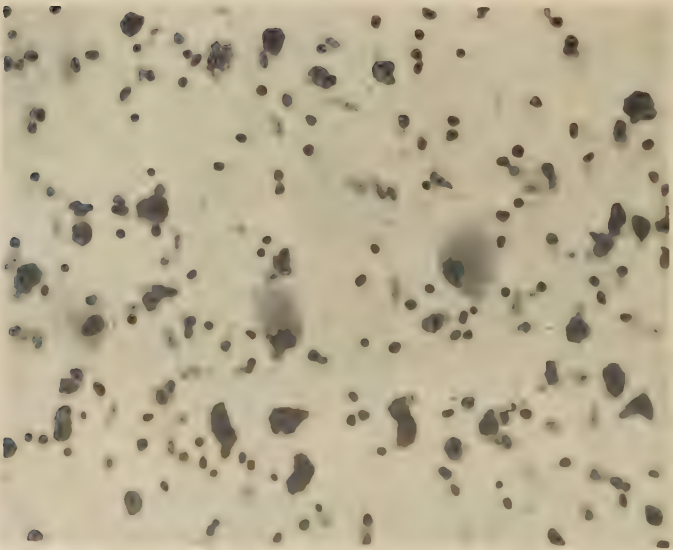


FIG. 4.—Cerebral cortex, postcentral gyrus: two distended pyramidal cells and numerous shrunken, coarsely granular nerve cells. Carbol azure $\times 240$.

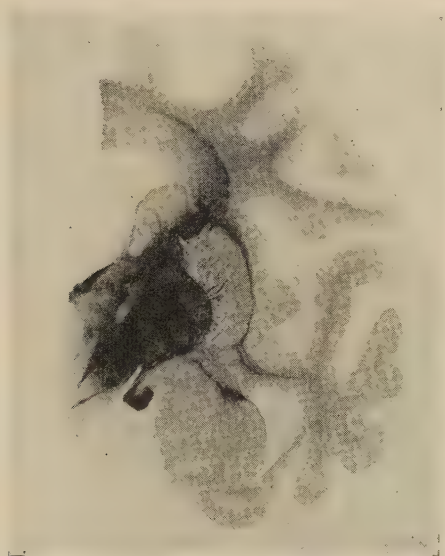


FIG. 5.—Coronal section of left hemisphere: demyelination of subcortical white matter. Kultschitsky-Pal $\times 1.2$.

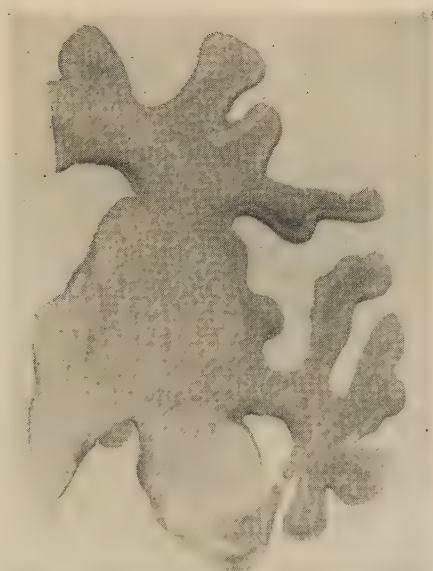


FIG. 6.—Coronal section of left hemisphere: gliosis of subcortical white matter. Holzer $\times 1.3$



FIG. 7.—Cerebellum: atrophy of granular layer with relative preservation of Purkinje cells.
Carbol azure $\times 30$.



FIG. 8.—Cerebellum: swelling of Purkinje cells and proliferation of Bergmann astrocytes.
Carbol azure $\times 240$.

myelinated, but some of the myelin sheaths are swollen and beaded. It also shows heavy gliosis.

Brain stem: All nuclei show moderate lipid storage in their nerve cells without apparent cell loss. The cerebral peduncles and the long descending tracts in the pons are reduced in size, poorly myelinated and heavily gliosed. There is also marked marginal and periaqueductal gliosis. In the pons the tegmentum is well myelinated as are the transverse ponto-cerebellar fibres.

Cerebellum: The cortex both of the vermis and of the hemispheres shows atrophy of the granular layer with astrocytic and microglial proliferation (Fig. 7). The Purkinje cells have largely survived and show evidence of lipid storage both in the cell bodies and in the dendrites which show irregular fusiform swellings or antler-like formations (Fig. 8). There is heavy gliosis of the cortex where the Bergmann fibres are particularly prominent and also of the white cores of the folia where the myelination is well preserved. The dentate nucleus shows neuronal lipid storage, but no cell loss, demyelination or gliosis.

Spinal cord: Only the upper cervical segment was available for examination. Although most cells show some evidence of lipid storage the anterior horn cells contain healthy nuclei and Nissl bodies. The lateral pyramidal tracts are gliosed and poorly myelinated.

Histochemistry

All staining reactions were performed on frozen sections of the brain fixed in 10 per cent. formol saline. The results are summarized in Table I. Some of the reactions were repeated after extraction with cold alcohol and with cold chloroform-methanol and gave identical results. In addition a more limited series of tests was carried out on paraffin sections of the liver, spleen, lymph node, thymus and pituitary. The results did not differ substantially from those obtained on frozen sections of the brain.

TABLE I
Staining Reactions of the Stored Substance

Method	Result
Kultschitsky-Pal myelin method	Negative
after progressive mordanting (Hurst)	Negative
Baker's acid haematein	Trace
after pyridin extraction	Negative
Scharlach R (Herxheimer)	Orange
Jackson's modification	Orange-yellow
Sudan black B	Bluish-black
Nile blue sulphate	Blue
Copper phthalocyanine (luxol fast blue)	Light blue
after ribonuclease	Unchanged
Okamoto's reaction	Positive
Periodic acid-Schiff	Strongly positive
Feyrter's thionin inclusion method	No metachromasia
Coupled tetrazonium	Reddish-brown

CHEMISTRY

Methods

The methods of lipid analysis are those employed in a previous paper (Tingey, 1956) with the exception of the ganglioside.

Ganglioside: Estimations were carried out both of neuraminic acid and of hexosamine. Neuraminic acid was estimated on the dried material by the method of Klenk and Langerbeins (1941), but owing to the difficulty in obtaining adequate neuraminic acid standards and to the fact that quantitative estimation is not always possible in the white matter, the results have been expressed semi-quantitatively (Nil=N.A. absent or not estimable, +=present in approximately normal quantities, ++=present in moderate excess, +++=present in gross excess).

Hexosamine was estimated on aliquots of the lipid extract after the method of Blix (1948). Hexosamine is also a component of substances other than ganglioside, but these are not normally soluble in lipid solvents. It is therefore reasonable to assume that variations in the lipid hexosamine content reflect fairly accurately the variations of ganglioside level (Smits and Edgar, 1957).

Cerebroside: It has been customary to calculate total cerebroside by multiplying the total lipid galactose by 4.55. This, however, is based on two erroneous assumptions; one that the ganglioside forms part of the cerebroside, and the other that both substances have the same galactose content. We have therefore adopted the procedure of Edgar (1955, 1956) of subtracting the ganglioside galactose from the total lipid galactose and multiplying the difference by 4.55. As some theoretical objections have been raised against this procedure (Svennerholm, 1956) the actual figures obtained for lipid hexosamine and lipid galactose are presented in Table III, which also serves to show the percentage of ganglioside galactose of the total lipid galactose.

Sphingomyelin: The usual method of estimating this substance by difference between the total lipid phosphorus and the "KOH splittable" phosphorus is open to the objection that it will include Cephalin B (Brante, 1949). An alternative way which does not include Cephalin B is to assess the sphingomyelin by difference between the total lipid choline and the "KOH splittable" choline (Brante, 1949), and figures for sphingomyelin given in our tables are derived in this manner. In order to assess the Cephalin B we have also applied the former method: thus (sphingomyelin+Cephalin B) will represent the sphingomyelin as it is more usually given in the literature.

Effect of Formalin: Formalin fixation leads to a reduction in phosphatides, particularly Cephalin A; but does not substantially affect either sphingomyelin, glycolipids or sterols (Brante, 1949). It may lead to high values for sphingomyelin by rendering some of the phosphorus non-releasable by the KOH (Schmidt *et al.*, 1946).

A low sphingomyelin value, however, may still be taken as evidence of demyelination, as the effect of the formol, if any, will be to raise the sphingomyelin value. The white matter of a case estimated by us which had been ten years in formol showed a large phosphatide reduction almost entirely at the expense of the Cephalin A.

Controls

The present case aged 4 years 8 months (one year in formol) was compared with a normal child aged 5 years (2½ years in formol). The juvenile A.F.I. case aged 24 (23 days in formol) was compared with our average of five normal adults (fresh).

TABLE II
Cerebral Lipids in Grams per 100 g. Dry Tissue

Case	Cortex					White Matter				
	Present Case	Normal Child	Juvenile A.F.I.	Average Normal Adult (av. 5)	Tay-Sachs	Present Case	Normal Child	Juvenile A.F.I.	Average Normal Adult (av. 5)	Tay-Sachs
Time of fixation	1 year	2½ years	23 days	Fresh	Not known	1 year	2½ years	23 days	Fresh	Not known
Total lipid ..	14.3	31.0	36.8	34.9		22.3	58.0	50.2	60.3	31.4
Cholesterol ..	3.5	5.7	6.9	6.5		4.9	14.4	9.0	12.9	7.5
Cholesterol ester ..	nil	nil	0.6	0.6		0.7	0.6	0.8	0.2	nil
Cerebroside ..	0.34	3.45	3.35	2.76	0.23	2.35	10.40	7.55	12.00	1.60
Ganglioside ..	0.74	1.80	1.39	1.67	9.30	0.42	0.32	0.26	0.51	7.50
(hexosamine)										
Ganglioside ..	+	+	+	+		+	nil	nil	nil	+++
(neuraminic acid)										
Phosphatide ..	8.9	17.6	18.2	19.4		15.0	19.7	20.0	25.3	6.9
Lecithin ..	3.5	7.9	8.4	5.5		7.8	7.3	7.2	7.5	2.4
Sphingomyelin ..	1.0	1.5	1.9	2.4		1.4	3.0	2.1	2.7	1.3
Cephalin A ..	2.9	5.1	6.0	7.9		5.3	4.5	7.1	11.3	1.6
Cephalin B ..	0.5	3.1	1.9	3.6		0.5	4.9	3.6	3.8	1.6
Unidentified lipid	0.82	2.45	6.96	4.57		nil	13.18	13.39	9.59	7.9

TABLE III
Hexosamine and Galactose Values in Grams per 100 g. Dry Tissue

Case	Cortex				White Matter			
	Average Normal (6 cases)	Present Case	Juvenile A.F.I.	Tay-Sachs	Average Normal (6 cases)	Present Case	Juvenile A.F.I.	Tay-Sachs
Lipid hexosamine	·174	·09	·168	1·12	·050	·050	·031	·890
Ganglioside galactose	·58	·30	·56	3·75	·166	·166	·104	3·00
Total lipid galactose	1·29	·375	1·30	3·80	2·52	·680	1·76	3·35
Percentage of ganglioside galactose in relation to total ..	45·0	80·0	43·0	98·0	6·6	24·4	5·9	89·5

TABLE IV
Myelin Constituents in Grams per 100 g. Dry Tissue

		Cortex				White Matter					
		Present Case	Normal 5 year old	Juvenile A.F.I.	Average Normal Adult	Tay-Sachs	Present Case	Normal 5 year old	Juvenile A.F.I.	Average Normal Adult	Tay-Sachs
Cholesterol ..	3·5	5·7	6·9	6·5	0·23	4·9	14·4	9·0	12·9	7·5	
Cerebroside ..	0·34	3·45	3·35	2·76		2·35	10·40	7·55	12·00	1·60	
Sphingomyelin ..	2·0	1·5	1·9	2·4		1·4	3·0	2·1	2·7	1·3	
Total myelin lipids	5·84	10·65	12·15	11·66		8·65	27·80	18·65	27·60	10·40	

DISCUSSION

Chemistry

Ganglioside has been found in considerably increased amounts in all cases of Tay-Sachs' disease, both in the cortex and in the white matter (Klenk, 1953). In the juvenile type no increase in the total amount of ganglioside has been found by the majority of workers (Klenk, 1953) with the exception of Cumings (1953) who found some increase in neuraminic acid in all types of amaurotic idiocy and of Diezel (1954a) who found an excess of neuraminic acid in a specially prepared concentrated cell extract. The findings of these authors, expressed semi-quantitatively, await further confirmation. The intracellular storage represents only one aspect of the profoundly deranged lipid metabolism in amaurotic idiocy and most cases exhibit other abnormalities in the concentration and distribution of cerebral lipids which are as yet poorly understood and difficult to interpret.

The results of the chemical analysis in the present case are decisive for its inclusion in the Batten group. Not only does it not reveal the great accumulation of ganglioside typical of Tay-Sachs' disease, but the amount of this substance is actually reduced. If, however, allowance is made for the severe destruction of the cerebral cortex, the amount of ganglioside present is probably within the normal range for the reduced cell population and is in keeping with the findings in other cases of Batten's disease. This does not necessarily mean that the substance stored in Batten's disease is not ganglioside, since the accumulation of this substance within the cell body may be due to faulty distribution and not to overall excess. We should also like to draw attention to two features which are sometimes quoted in support of the diagnosis of amaurotic idiocy. One is the relative increase of the amount of ganglioside in proportion to total glycolipid (see Table III), the other is the presence of neuraminic acid in the white matter. While both these points are obviously true in cases with gross excess of ganglioside, they may be fallacious in cases of severe demyelination. Any substantial reduction of myelin lipids will upset the cerebroside-ganglioside ratio in favour of the latter. Furthermore, the negative result of the Bial reaction in normal white matter is not due to absence of neuraminic acid, but to the fact that myelin lipids interfere with the reaction (Klenk and Langerbeins, 1941). Neuraminic acid thus becomes estimable when

myelin lipids are reduced or absent. The degree of demyelination in our case can be judged from Table IV in which the values of the myelin constituents are presented. Another interesting fact emerges from the study of these figures, namely, that there is a reduction in total myelin lipids in the white matter of the control case of juvenile amaurotic idiocy despite the apparently normal myelination as revealed by histological staining. It appears that chemical analysis can be a more sensitive gauge of minor degrees of diffuse demyelination than histological examination.

Histochemistry

At the present time no single test or series of tests is sufficiently specific to identify individual lipids, particularly if present in a mixture. In our case the histochemical properties of the stored substance resembled those generally found in the Batten group in the affinity to Sudan dyes, the insolubility in lipid solvents and the failure to stain with haematoxylin. These distinctions are not, however, absolute, for Globus (1923) found variable haematoxylin staining in his infantile cases while a positive result by this method was recorded in the juvenile cases of Rogalski (1910) and Schob (1912). Moreover, the results of extraction must be interpreted with great caution when fixed material is used. The value of the more recently evolved histochemical tests (Diezel, 1954b) is difficult to assess and more cases need to be examined. It is, however, worth noting that the results in our case corresponded closely to those found in the juvenile control except that in the latter the stored substance stained more intensely with luxol fast blue.

Cerebral Pathology

In the present case the moderate distension of the nerve cell bodies with lipid and the absence of dendritic expansions except in the cerebellum were features shared in common with the juvenile form. On the other hand, the widespread loss of neurones in the cerebral cortex was more reminiscent of the findings in cases of Tay-Sachs' disease in which life has been unusually protracted (Aronson *et al.*, 1955). This tendency to neuronal destruction is a striking feature of most of the early and more rapidly progressive cases of the Batten group. It would appear that in these subacute cases an unusually severe metabolic disturbance has led to the early death of many cells. In the chronic, or classical juvenile form of the disease, the slow accumulation of lipid does not seem to embarrass the nerve cell unduly for even in the more distended cells the nucleus usually appears healthy and the neurofibrils are displaced rather than destroyed. A similar situation occurs in infantile Gaucher's disease (Norman, Urich and Lloyd, 1956) where a distinction can be drawn between a milder process leading only to the storage of lipid and a severe metabolic disturbance causing degeneration and death of nerve cells.

Another peculiar feature of some of these subacute cases is the presence of large numbers of deeply staining, rounded and coarsely granular cells with pyknotic nuclei. In our case many of these closely resemble phagocytic microglia though there is little doubt as to their neural origin. These cells are not found in the molecular layer or subcortical tissue, they show no tendency to aggregate around blood vessels and their granules have the same staining properties and insolubility as the material found in nerve cells in other parts of the brain. Transitional forms can also be traced between these shrunken cells and the more obvious neurones.

As in many other of the late infantile cases reported in the literature the myelination of the central white matter was notably defective. This feature is shared with Tay-Sachs' disease and may in part be due to the relatively early onset of the disease in both these variants. In this connection it is of interest to note that none of the late infantile cases of the Batten group (see Appendix) has shown the widespread status spongiosus with a grossly enlarged brain occasionally found in Tay-Sachs' disease (Steegman and Karnosh, 1936). A small, atrophic cerebrum with generalized gliosis of the white matter is usual.

The cerebellar atrophy in the present case is of the "cerebello-petal" type (Bielschowsky, 1920), commonly found in the juvenile form of the disease, that is to say, there is a loss of granule cells and disappearance of climbing, mossy and basket fibres with relative preservation of the Purkinje cells. This pattern of lesions is not always present throughout the whole cerebellum. In Haddenbrock's (1950) case, for example, which belonged to the Batten type of late infantile amaurotic idiocy, the hemispheres showed atrophy of the granular layer while in the vermis the Purkinje cells were mainly affected. In Tay-Sachs' disease severe cerebellar atrophy may also be present (Holmes, 1911) but the whole cortex is involved and the appearance of the less affected areas suggests no special predilection for the granule cells.

SUMMARY

The chemical and pathological findings in a case of late infantile amaurotic idiocy of the Batten type have been reported. Chemical analysis showed considerable reduction in the total lipid content of the brain, the myelin constituents being particularly affected. The ganglioside content was not increased. These results have been compared with those in Tay-Sachs' disease, juvenile amaurotic family idiocy and normal brains of various ages. The main pathological features of the condition, apart from lipid storage in the neurones, were severe atrophy of the cerebral cortex with marked shrinkage of many of the nerve cells and considerable reduction in their number, demyelination and gliosis of the central white matter and degeneration of the granular layer of the cerebellum. Microscopical evidence of lipid storage was also found in the cells of the reticulo-endothelial system. A review of previously reported cases of late infantile onset shows that the majority belongs to the Batten group and these cases constitute a subacute and precocious variant of the classical juvenile form of amaurotic idiocy.

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APPENDIX

Review of the Cases of Late Infantile Amaurotic Idiocy Belonging either to the Tay-Sachs' or Batten Group

The cases of late infantile onset which properly belong to Tay-Sachs' group are those which show the cherry-red spot (Wolfssohn, 1915; Cordes and Horner, 1929; Greenfield and Nevin, 1933) and also those whose siblings were affected by the typical Tay-Sachs' syndrome (Higier, 1906). The cases of Hassin and Parmelee (1928) and of Jervis (1940) almost certainly belong to this group in spite of the absence of typical eye changes and of a family history as they resemble infantile cases in all other features.

The cases of late infantile onset belonging to the Batten group of which we have pathological accounts are those of Jansky (1908), Bielschowsky (1913-14), Brodmann (1914), Batten and Mayou (1915), Torrance (1922), Frets and Overbosch (1923), Hassin (1926), Richter and Parmelee (1935), Bielschowsky (1936), Haddenbrock (1950) and Greenfield, Aring and Landing (1955). To these may be added cases of which only clinical and ophthalmological accounts are available: Batten (1903, Case 2), Mülberger (1903), Ichikawa (1909), Wyburn-Mason (1943, Case 11). The cases of Marinesco (1930) and of Schlesinger, Greenfield and Stern (1934) showed optic atrophy without pigmentary changes, and in spite of some atypical features probably belong to the Batten group.

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THE PROBLEM OF UNEMPLOYMENT AMONG EPILEPTICS

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INTRODUCTION

WHEN the diagnosis of epilepsy is made, the patient must first of all be investigated to make sure that the epilepsy is not symptomatic of a disease that will need some special line of treatment. For example, epilepsy starting at any age may be the first indication of a cerebral tumour, although this will obviously be more likely among the older patients. The nature and extent of these investigations will depend on such factors as the age of onset, the type of seizure and the associated positive physical signs. However, even in the cases where there is no evidence whatsoever that the epilepsy is symptomatic, an electroencephalogram may help in differentiating the type of epilepsy from which any particular patient is suffering, and so aid in deciding on the form of treatment most likely to be beneficial. When any necessary investigations have been carried out and the form that treatment is to take has been settled, the social implication of this diagnosis has then to be faced. This often resolves itself into a discussion of the patient's employment.

Many factors will have to be taken into account, such as the length of time the patient has suffered from fits, their frequency and the particular form they take. If the diagnosis has just been made the patient may naturally be worried about his job; if he will be able to keep it, and if it is suitable for anyone liable to sudden attacks of loss of consciousness. On the other hand, if the patient has had fits for many years, he may come for advice after repeatedly losing jobs in spite of an expressed desire to work.

In view of this recurring problem of the employment of epileptic patients, it was decided to enquire into it more fully and to see if the impressions gained by casual questioning of individual patients were confirmed by a more detailed analysis. In particular, it seemed justifiable to find out if the problem was one of such magnitude as it might be expected to be. Obviously any study of this kind undertaken at the present time would have to be reviewed against a background of full employment or more exactly of a situation where there are often more jobs than people to fill them.

Four hundred patients were chosen at random from among those attending the Out-Patient Department at the National Hospital, Queen Square. A hundred of these were women between the ages of fifteen and sixty, two hundred were men between the ages of twenty-six and forty-five, fifty were men below the age of twenty-six and fifty were men above the age of forty-five. The occupations of these patients were graded in the following manner:

- A. Technical and scientific, e.g., schoolteacher.
- B. Administrative, e.g., inspector.

- C. Commercial, e.g., clerks, typists, secretaries, telephonists.
- D. Skilled factory, e.g., machining, tailoring, dressmaking, assembling, book-binding, upholstering.
- E. Unskilled factory, e.g., packing, boxmaking, sorting.
- F. Manual, e.g., labourer for local council.
- G. Personal service, e.g., saleswoman, shop assistant, housekeeper, kitchen and domestic worker, waitress, nurse, cinema usherette.

The epileptic attacks from which these patients suffered were classified under the following headings: grand mal, petit mal, fits arising in the temporal lobes, other focal epileptic fits, and minor fits of unknown origin. The term petit mal in this paper has been used to refer to brief interruptions of consciousness associated with outbursts of generalized three per second spike and wave complexes in the electroencephalogram. When such attacks were associated with a normal electroencephalogram or with only non-specific changes, they were usually classified as of unknown origin because clinically such seizures as the "pseudo-lapses" of temporal lobe epilepsy may be difficult to distinguish from petit mal. However, occasionally the clinical history left no reasonable doubt that the attacks were of diencephalic origin in spite of a negative electroencephalogram.

It also seemed important to estimate the level of intelligence of these patients, as it was realized that this would undoubtedly influence the type of their employment, regardless of whether they suffered from epilepsy or not. Information was also wanted on the possible relationship between the level of the I.Q. and the type of epilepsy from which the patient suffered. An estimate was made of the I.Q. on the basis of the patients' scores in two subtests of the Wechsler scale, namely vocabulary and digit symbol. The patients were then allotted to one of the following groups:

1. Below Average, I.Q. less than 93.
2. Average, I.Q. 93 to 107.
3. Above Average, I.Q. greater than 107.

It should be stated that a patient who only kept a job for a very short period and was frequently without one was regarded for the purposes of this review as unemployed. It seemed important to make this adjustment as on the day of interview a particular patient might have just obtained work although at no time in the past could he really be considered as economically employed.

ANALYSIS OF RESULTS OF INTERVIEWS WITH 400 EPILEPTIC PATIENTS

In order to avoid repetition the patients will be referred to under the following groups:

- Group 1. Women between the ages of fifteen and sixty.
- Group 2. Men between the ages of twenty-six and forty-five.
- Group 3. Men below the age of twenty-six.
- Group 4. Men over the age of forty-five.

As the majority of male patients were between the ages of twenty-six and forty-five, it seemed justifiable to consider them as a separate group. Men between these ages would obviously be at the height of their earning capacity as long as their health remained good, and it was considered important to find out how they fared once they had been labelled as epileptics. The remaining males above and below these ages were then gathered together and

their clinical features analysed to see if there were any differences in the three groups that would indicate that age itself played a significant role in the employment of epileptics.

All the patients, both male and female, were seeking gainful employment, and the percentages of those unemployed will be seen in Table I.

TABLE I

Groups										Percentages Unemployed
1	..	..	..	..	..	..	..	..	..	9
2	..	..	..	..	..	..	..	..	..	6·5
3	..	..	..	..	..	..	..	..	..	10
4	..	..	..	..	..	..	..	..	..	18

The relatively few epileptic patients who were unable to find work was the most striking finding in this survey, and one that restricted the conclusions that could be drawn from it. It might have been expected that the older epileptic patient would find it particularly difficult to obtain a suitable job and this may account for the higher percentage of unemployed in Group 4.

The distribution of the occupational grades among the patients in each of the four groups will be seen in Figures 1 to 4. The largest number of women had found employment as clerks or domestic workers, or in closely allied jobs.

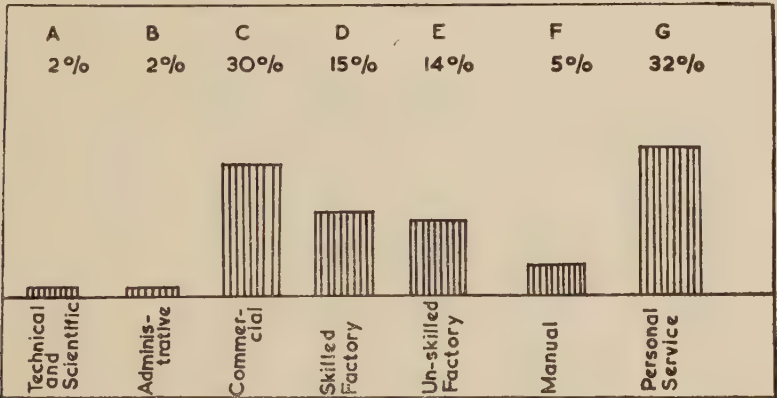


FIG. 1.—Distribution of occupations amongst 100 adult epileptic females.

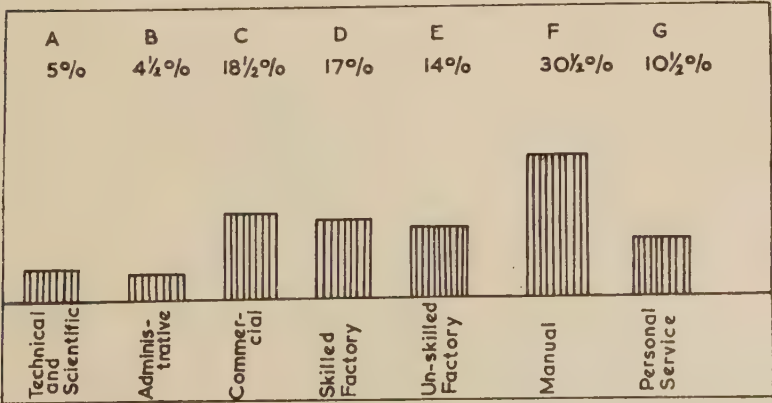


FIG. 2.—Distribution of occupations amongst 200 epileptic males between 26 and 45 years old.

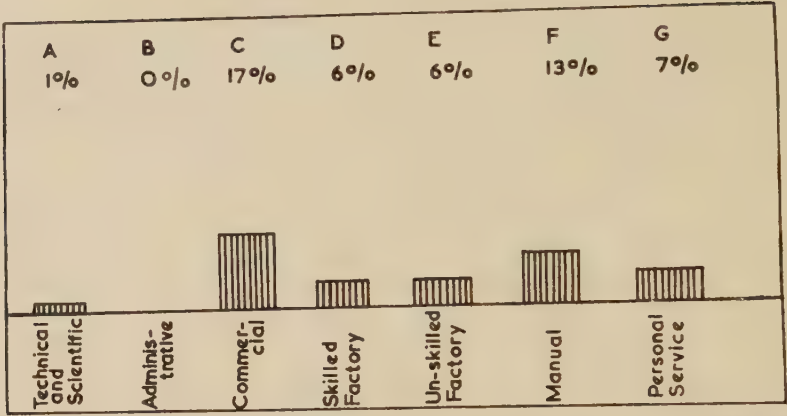


FIG. 3.—Distribution of occupations amongst 50 epileptic males under 26 years old.

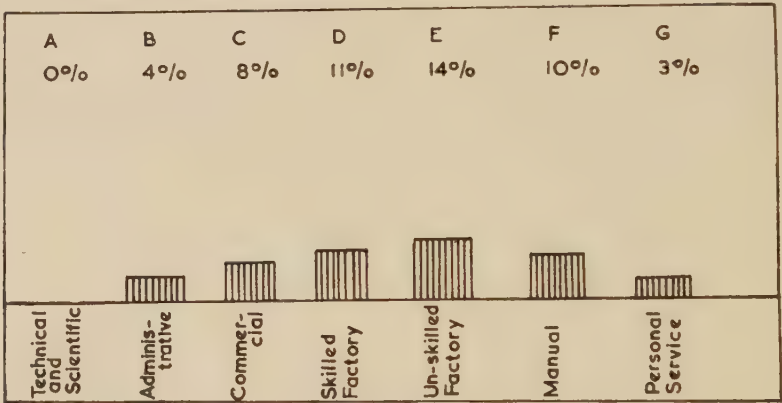


FIG. 4.—Distribution of occupations amongst 50 epileptic males over 45 years old.

Most of the men in Group 2 were manual workers.

It was difficult to be certain how many of these patients had told their employers that they suffered from epilepsy, especially as, even if the patients thought that their disability was unknown at their place of work, information about this might have been obtained from other sources. However, the percentages of patients in the various groups who definitely stated that their employers knew of their epilepsy were ninety-two, sixty, forty-four and forty-two. It is possible that men were more reluctant than women to admit that they had seizures because the competition for their jobs was greater, and the results of unemployment more far-reaching, at least for those with dependent families.

Table II gives details of the presence or absence of a warning at the onset of an attack of unconsciousness sufficient to enable the patient to take precautionary measures.

TABLE II
Presence or Absence of a Significant Aura

	Group 1	Group 2	Group 3	Group 4
Percentage of patients who always had warning of fits	8	27	26	18
Percentage of patients who sometimes had warning of fits	11	12	8	10
Percentage of patients who never had warning of fits	81	61	66	72

Again the men tended to play down the effects of their attacks on their employability, and this fact has to be taken into account when assessing these figures. An impression was gained that a warning that the patient considered would remove any risk to himself or others, should he lose consciousness, was not always so in practice. It is doubtful if the patients seriously considered the fact of such a warning occurring when deciding whether to tell their employers of their attacks, although in Group 2, for instance, the percentage of patients with no adequate warning was higher among those who did not. Certainly if the patient was able to take precautionary measures before losing consciousness, it would lessen the disability of his attacks, and, therefore, he might be less inclined to conceal them.

Figures 5 to 8 show the distribution of the various types of fits among these patients, the majority suffering from grand mal, either alone or in com-

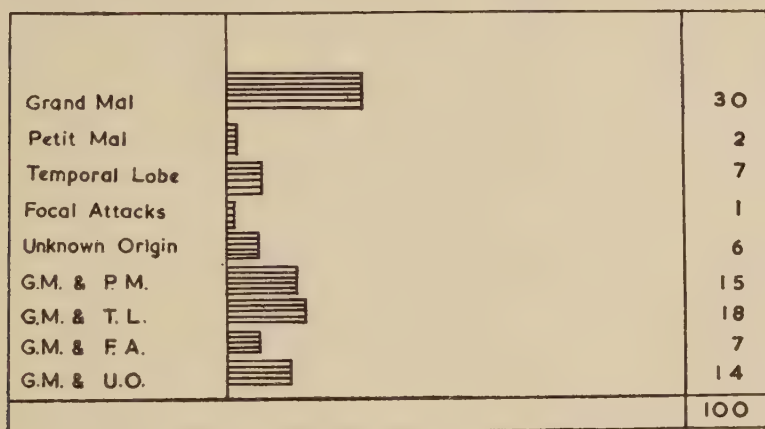


FIG. 5.—Type of attacks suffered by 100 adult epileptic females.

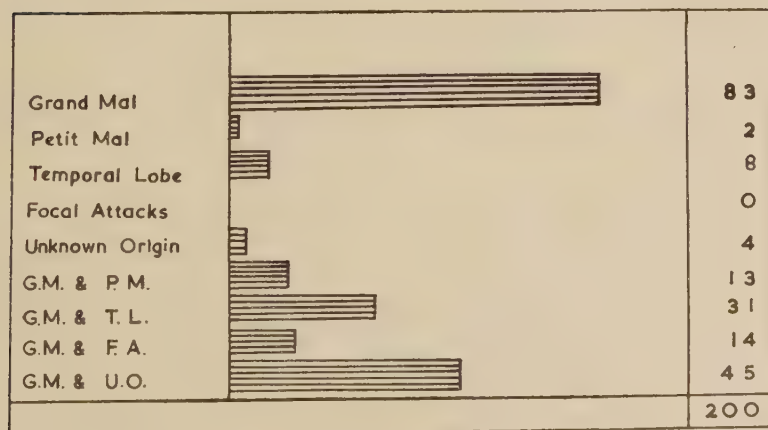


FIG. 6.—Type of attacks suffered by 200 epileptic males between 26 and 45 years old.

bination with minor seizures. When the incidence of petit mal is compared in Groups 2, 3 and 4, it will be seen that, as might be expected, these attacks become increasingly rare in the older age groups. Even in Group 3, where they are most frequent, they are almost all associated with grand mal, suggesting

Grand Mal		14
Petit Mal		2
Temporal Lobe		1
Focal Attacks		2
Unknown Origin		1
G.M. & P.M.		9
G.M. & T.L.		4
G.M. & F.A.		8
G.M. & U.O.		9
		50

FIG. 7.—Type of attacks suffered by 50 epileptic males under 26 years old.

Grand Mal		11
Petit Mal		0
Temporal Lobe		1
Focal Attacks		1
Unknown Origin		2
G.M. & P.M.		2
G.M. & T.L.		13
G.M. & F.A.		6
G.M. & U.O.		14
		50

FIG. 8.—Type of attacks suffered by 50 epileptic males over 45 years old.

that, as the patient grows older, these minor attacks either stop or become combined with major fits. In Group 4, there was a considerably higher percentage of patients with grand mal combined with minor seizures arising in the temporal lobes, than of patients with this variety of minor epilepsy alone. This finding confirms the tendency for patients with temporal lobe epilepsy to suffer only from focal fits for a number of years, and then, as they grow older, to have generalized ones as well.

The type of epileptic attack in relation to the grade of employment has also been analysed (Figs. 9 to 12), and this shows that there is no significant preponderance of any particular variety of epilepsy among any one grade.

The timing of the fits was then considered to see if this had any effect on the patients' ability to obtain employment. Table III shows the percentage of patients, both employed and unemployed, who had the attacks only at night, or only during the day, or during both.

Grade	G.M.	P.M.	T.L.	F.A.	U.O.	G.M. P.M.	G.M. T.L.	G.M. F.A.	G.M. U.O.	Total
A	..			1		1				2
B	..						1		1	2
C	..	8	1	3	2	3	7	3	5	32
D	..	8	1	1	1	2	2		3	18
E	..	5	1		1	2			1	10
F	..	1		1			1			3
G	..	8	2		2	8	5	4	4	33
	30	3	7	1	6	16	16	7	14	100

FIG. 9.—Type of attacks in relation to grade of employment of 100 adult epileptic females.

Grade	G.M.	P.M.	T.L.	F.A.	U.O.	G.M. P.M.	G.M. T.L.	G.M. F.A.	G.M. U.O.	Total
A	..	4			1	1	2	1	1	10
B	..	4				1	2		2	9
C	..	20		1		3	6	2	5	37
D	..	14	1	1	1	2	3	3	9	34
E	..	10		1	2	1	4	1	9	28
F	..	24		4	1	3	11	4	14	61
G	..	7	1			2	3	3	5	21
	83	2	8	0	4	13	31	14	45	200

FIG. 10.—Type of attacks in relation to grade of employment of 200 epileptic males between 26 and 45 years old.

Grade	G.M.	P.M.	T.L.	F.A.	U.O.	G.M. P.M.	G.M. T.L.	G.M. F.A.	G.M. U.O.	Total
A	..							1		1
B	..									0
C	..	3	1		1	4	2		5	17
D	..	4				1		1		6
E	..	1	1				2		1	6
F	..	4		1		3		3	2	13
G	..	2				1		3	1	7
	14	2	1	2	1	9	4	8	9	50

FIG. 11.—Type of attacks in relation to grade of employment of 50 epileptic males under 26 years old.

Grade	G.M.	P.M.	T.L.	F.A.	U.O.	G.M. P.M.	G.M. T.L.	G.M. F.A.	G.M. U.O.	Total
A	..									0
B	..			1			1		2	4
C	..		1		1		3		3	8
D	..	3					2	4	2	11
E	..	3				1	6	2	2	14
F	..	4			1	1	1		3	10
G	..	1							2	3
	11	0	1	1	2	2	13	6	14	50

FIG. 12.—Type of attacks in relation to grade of employment of 50 epileptic males over 45 years old.

TABLE III

Percentage of Patients in Various Groups

Timing of Epileptic Fits											
				Grade 1		Grade 2		Grade 3		Grade 4	
				Em- ployed	Unem- ployed	Em- ployed	Unem- ployed	Em- ployed	Unem- ployed	Em- ployed	Unem- ployed
Nocturnal	..	..	..	11	3	10	0.5	4	0	16	0
Diurnal	..	..	..	25	3	30	1.5	34	2	26	2
Both	..	..	..	55	3	52	4.5	44	8	40	10
No information	..	..	..	0	0	1.5	0	8	0	0	6

These figures do not suggest that it mattered when a patient had his attacks, and, as far as obtaining a job was concerned, the fact that such a diagnosis as epilepsy had been made was probably of more importance.

The level of intelligence of these patients was roughly assessed by the method already discussed. The results of this examination in each group will be seen in Table IV.

TABLE IV

Assessment of Intelligence

Levels of Intelligence					Percentage of Patients in Various Groups			
					Group 1	Group 2	Group 3	Group 4
Above average	..	..	..	..	20	21½	16	18
Average	..	..	..	..	59	55½	38	50
Below average	..	..	..	..	21	22	36	28
No information	..	..	..	..	0	1	10	4

The figures are very similar in each of the groups except for the larger number of men below the age of twenty-six who were of a low level of intelligence.

The types of epilepsy from which the patients in these various intelligence levels suffered has been analysed, but it was found that no particular type was associated with either a high or low intelligence.

The clinical features of the few unemployed patients were then considered. Table V gives the variety of seizures suffered by these men and women.

TABLE V

The Types of Epilepsy Diagnosed in the Unemployed Patients

					Group 1	Group 2	Group 3	Group 4
Grand mal	..	..	..	..	1	3	1	4
Petit mal	..	..	..	..	—	—	—	—
Temporal lobe	..	..	..	..	—	—	—	—
Focal attack	..	..	..	..	—	—	—	—
Unknown origin	..	..	..	..	—	—	1	—
Grand mal and petit mal	..	..	..	..	5	2	1	1
Grand mal and temporal lobe	..	..	..	..	2	—	—	2
Grand mal and focal attack	..	..	..	..	1	1	2	1
Grand mal and unknown origin	..	..	..	..	—	7	—	1

All but one of the unemployed patients had grand mal, either alone or combined with minor fits of one kind or another, and it certainly seemed to these patients that the major attacks were the cause of their inability to keep their jobs for any length of time.

The unemployed often had frequent fits, some of them as many as fifty to a hundred or more grand mal during the year. However, although the majority of the patients in employment were not having so many fits, there were several who were having more, both major or minor, so that frequency alone was not a bar to gainful employment. The fits among the unemployed, compared with those among the employed, did not on the average last longer, and did not occur significantly more often without any warning. It was also found that the duration of the disease in those unemployed was much the same as for the groups as a whole, so that, apparently, a long history of epileptic attacks

did not influence the patient's ability to obtain suitable work. For instance, in Group 4, where the patients were likely to have suffered from fits for the longest periods and therefore, where any differences would be most apparent, the average duration among the unemployed was thirty years, and among the employed was twenty-eight. The finding of most significance in the study of the unemployed was the number who were of below average intelligence. In Table VI will be seen the number of patients in each group of such low intelligence.

TABLE VI
Number of Patients of Below Average Intelligence

					Group 1	Group 2	Group 3	Group 4
Unemployed	..	..	..	..	7 (9)	7 (13)	3 (8)	6 (9)
Employed	..	..	..	..	14 (91)	15 (187)	15 (45)	8 (41)

(The numbers in brackets indicate the total number of patients unemployed and employed in each of the four groups.)

It is apparent from these figures that the patient's level of intelligence may well have been of more importance as far as holding down a job was concerned than the fact that they suffered from epilepsy. Finally, although an occasional patient among the unemployed complained that they were unduly sleepy, there was no evidence that any of them were taking an excessive dose of any single drug or combination of drugs.

On comparison of the findings in Groups 2, 3 and 4, there were no differences between them of such significance as to suggest that the age of a male patient suffering from epilepsy affected his chances of obtaining economic employment, taking into account that it will always be slightly more difficult to obtain work as age increases.

DISCUSSION

The information reviewed in this paper was obtained after the patients had been interviewed by both doctor and social worker. The interviews had, of necessity, to be brief, but it seemed preferable to concentrate on a relatively few selected questions designed to elicit particular points of information so that as large a number of patients as possible could be included. This also meant that the interview could be standardized so that the answers to the various questions would be comparable when the time came to analyse the results. When the investigation was started, it had been expected that there would be a large number of unemployed among both men and women suffering from epilepsy, in spite of the fact that it was a time of full employment. It was therefore a considerable surprise to find the figure was so low and it made it obvious that the nature of the problem was not such as had been imagined when the enquiry was begun. If so few epileptics were unable to obtain work, it seemed reasonable to assume that epilepsy alone was very seldom a bar to employment during the period under review. The number of the unemployed who were below-average intelligence made it clear that there were indeed other factors of importance in deciding whether these epileptic patients were able to hold down a job. This was supported by an analysis of another group of patients seen at the National Hospital. These were men and women who were referred directly to the hospital from the Labour Exchanges because it had proved impossible to settle them in suitable employment. It was found, except in a few of these patients who were having very frequent epileptic attacks, that the reason for

their unemployment was either a low level of intelligence or a disorder of personality which made it difficult or impossible for them to adapt to their varied circumstances. This makes it particularly important to assess carefully from the point of view of their intelligence and personality traits any epileptic patient who is having difficulty in finding work.

It was of interest to see that such factors as the duration of the disease and the type of epileptic attack from which the patient suffered had so little effect on employability. This again stresses that when deciding on suitable employment for these patients attention must not be confined to the various restrictions imposed by the nature of the fits from which any particular individual may be suffering. There will be the occasional patient, however, who cannot keep a job owing to the frequency of his attacks and the resultant time that is lost at work. It is sometimes found that these patients are not receiving as intensive treatment as they might be, so that an increased dosage of the tablets they were already taking, or a trial of alternative therapy may have to be considered. Also, although there will always be a small number of epileptic patients whose attacks cannot be controlled by drugs, the fact that the treatment is ineffective should always raise the possibility that the epilepsy is symptomatic. There is therefore an argument for admitting such patients to hospital for full investigation in case the reason for the poor response to treatment is the presence of some underlying cerebral disease. If this was found to be a focal lesion it might well be amenable to surgery.

There was no evidence among these patients that the treatment they were receiving significantly influenced their ability to obtain and hold a job. However, it is necessary to review this point from time to time as, particularly when a patient is on large and continuous doses of barbiturates, his efficiency may be seriously impaired. He will most likely complain of sleepiness and lack of concentration, but the change may be a more subtle one and affect the speed of his mental processes so that in consequence his work will suffer. It may be possible to counteract these symptoms by employing alternative drugs or combining his present ones with amphetamine sulphate. More obvious toxic symptoms need not be considered here, as they will quickly bring the patient under medical observation and stopping the incriminating drug usually restores the patient to normal health.

The medical treatment of these patients is not all that can be done for them. Many social problems may have to be faced, and help given in solving them. Some of these will be connected with the patient's family relationships and will include advice on such matters as marriage and children. However, if the patient is a wage earner the aspect of most importance to him may concern employment. As has been stated, this problem presents itself in two forms. If the diagnosis of epilepsy has just been made it may be necessary to advise the patient to change his job, should he, for example, be a driver of commercial vehicles. This may be a hard decision to make, as it often means a reduction in wages, anyhow for a while.

However, the outlook for the economic employment of these people is seldom nearly as difficult as they imagine, or as it might be expected to be, and the figures produced by the review of patients given in this paper are reassuring. It will naturally be a comfort to them to learn that so many suffering from a similar disability are able to hold down jobs, even if they are not ones they would choose under normal circumstances.

On the other hand, if the patients had been known to suffer from epilepsy for a long time, the problem will be slightly different. It will have been seen

from this review that the majority of epileptics do not have any great difficulty in obtaining work, and it has been its chief finding that the fits as such are very seldom the cause of unemployment. Some of these patients will therefore be among that small section of the community who are virtually unemployable by reason of their low intelligence or defective personality. However, a fair proportion of them can certainly be rehabilitated, especially if no one has previously attempted to help them in this way.

In any case, when an epileptic patient is referred to an almoner because it is thought advisable for him to change his job, or because he is having difficulty in obtaining suitable employment, a frank discussion of the situation will do much to reassure him. Advice can be given on the type of employment best suited to anyone liable to attacks of unconsciousness, and if the attacks are occurring with any degree of frequency, it will probably be best to place the patient's name on the Disabled Register. The patient may complain that this will inevitably lead to his or her employer knowing that they suffer from fits, but if these have been occurring more than occasionally during the day, it is inevitable that their disability will be found out, and then they may be treated with scant sympathy for their lack of frankness when applying for the job. It should therefore be pointed out to these patients that it may be easier to obtain work if no mention is made of epilepsy, but if they expect to keep in continuous employment for any length of time, it will be better not to hide the fact that they do suffer from this complaint. Then if their seizures are at all frequent it should undoubtedly assist them if they are on the Disabled Register, as firms employing more than twenty people have to have among their employees three per cent. who are so registered. Also if they are not registered, they may well be sent to unsuitable jobs by the Labour Exchange, so that they either have to give them up immediately or risk danger to themselves or others. The fact that an epileptic patient is debarred from certain occupations may well mean a reduction in earning power, and some of these patients may feel that their name being on the Disabled Register is the reason they are unable to get a better job. However, it should be pointed out that if their epilepsy had not been considered severe enough to hamper them obtaining satisfactory employment, such registration would not have been advised, and its sole object was to place them in a more advantageous position in the competitive labour market. It should also be explained that it may well be only a temporary measure and should their condition improve their name can then be removed from the Register. However, in the long run the benefit of this scheme to the patient depends on the help of the Disablement Resettlement Officer, and the almoner should keep in close touch with him to explain the particular needs and difficulties of the epileptic patient.

The recent Ministry of Health report on the medical care of epileptics stressed the difficulties of finding suitable jobs for these patients. Fellow workers may dislike the interruptions and upsets caused by seizures occurring at work and their employers may fear the consequent fall in production. The latter may also be concerned over the legal liability in case of accidents at work. However, provided reasonable precautions are taken there is no special liability for accidents to epileptics, and the National Insurance Act covers these patients in the same way as all other workers. The report also stresses the need for complete frankness with the employer regarding an epileptic's condition. This is of importance not only in the placing of an individual epileptic in suitable employment but also in the future relationship with a particular employer, who may well refuse to give work to similar patients again if one whom he has

accepted proves to be more prone to fits than he had been led to expect.

In conclusion it may be stated that although this review has shown that unemployment among epileptics is not the problem that it was expected to be, there is still much that can be done for the individual patient. Unfortunately the diagnosis of epilepsy still carries with it an unjustifiable stigma in the minds of the general public which is undoubtedly the result of ignorance. Much is being done by such organizations as the British Epilepsy Association to educate the public, but until this bears fruit, anyone suffering from this disease will need the help of both doctor and social worker. The former must give these patients an adequate explanation of their attacks, telling them that anyone may suffer from an epileptic fit but that some are more liable than others, and the fact that their threshold for such attacks is lower than the average may bear no relationship either to their mental or physical health. They should also be told how much can be done to help them by treatment of one kind or another, and they should be encouraged to ask questions on problems such as the hereditary nature of the disease. Every effort should then be made to see that these patients are suitably employed and they should realize that if they run into difficulties at any time they can come to the almoner for further advice and assistance. The lot of these patients would then undoubtedly be happier than it often is, and much unnecessary suffering would be avoided.

SUMMARY

Three hundred men and a hundred women suffering from epilepsy, who were employed or were seeking employment, were selected at random from among the patients attending the out-patient department at the National Hospital, Queen Square. The men were divided into three groups according to their age, and then these and the group containing the hundred women were analysed. The various features that it was thought might affect these patients' employability were considered in detail. These included, among others, the type of epilepsy from which the patients suffered; the relationship of these various types to the grade of employment; the time of occurrence of the fits; and whether there was a warning of the attacks of unconsciousness sufficient for reasonable precautionary measures to be taken. It was thought that the factor, other than epilepsy, that was most likely to influence these patients' ability to obtain work was their level of intelligence. A rough estimate was therefore made of this in the case of every patient, and they were classified as of above average, average, and below average intelligence.

The most remarkable feature of this survey was the few men and women who were not working. The vast majority of those who were working were economically employed, only an occasional patient being given a job in a family business which he might otherwise not have obtained, or working in such an organization as Remploy. The unemployed were considered in greater detail than the various groups as a whole, but their numbers were too small to allow any definite conclusions to be drawn. There were undoubtedly more patients with a below average intelligence among the unemployed than among those who were at work, and it became apparent that it was this factor and sometimes disorders of personality that more frequently prevented them obtaining work than the presence of a disability such as epilepsy.

Various problems connected with the employment of epileptic patients were then discussed, such as the necessity for some of these patients to change their jobs and the advisability for those who were having frequent attacks to be registered as disabled. It was stressed that although the problem of unemployment among epileptics is at the moment a small one, there was still much that could be done to help these patients by both the doctor and the social worker.

ACKNOWLEDGMENTS

The authors wish to thank the Medical Committee of the National Hospital, Queen Square, for permission to publish details from the case records of patients attending the out-patient department, and wish to acknowledge the invaluable help of Mrs. B. Johnstone in analysing the information obtained after interviewing the patients.

A SURVEY OF 44 CHILDREN ADMITTED TO THE FOUNTAIN GROUP HOSPITAL UNDER THE MENTAL DEFICIENCY ACT AND SUBSEQUENTLY ACCEPTED AS EDUCABLE

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I. INTRODUCTION

BETWEEN 1 January, 1948 and 1 July, 1956, forty-one children in the Fountain Group Hospital were accepted as educable by ten Education Authorities and they were transferred to appropriate schools. In addition three children under 5 years were found on admission to be of average intelligence. All forty-four children had been admitted to this hospital of approximately 700 beds for low-grade mentally defective children.

It was considered that the progress of these educable children would be of interest, and this survey presents the status of the children on 1 July, 1956. Two children have been excluded from this survey. One was a girl who, after 5 years and 9 months in this hospital, was accepted as Educationally Subnormal. She had been at school for 7 years by 1956, but on 1 July, 1956 she and her family could not be traced. It seems likely that she is still considered educable. The other, a boy with multiple handicaps, returned home to await a special school vacancy but he was admitted to a hospital unit for severely handicapped boys where some education was provided.

II. STATUS AT FOLLOW-UP

The children were transferred to twelve different schools which can be classified into four groups (Table I).

TABLE I

The Classification of the Schools to Which the Children were Transferred

Type of School	Completed Education	Still at School	Excluded as Ineducable	Not Included
E.S.N. (educationally subnormal)	5	9	8	1
E.S.N. and other handicaps ..	—	9	—	1
Other handicaps	—	7	1	—
Normal	—	3	—	—
Total (44)	5	28	9	2

The educationally sub-normal and other handicap group includes the schools providing for the additional handicap of blindness, partial sight, deafness and physical handicap. The other handicap group are the normal special

schools for the deaf; the blind; the physically handicapped; the epileptic and the maladjusted.

It is understandable that two-thirds of the 44 children were accepted as educationally subnormal, but it is interesting that the ten receiving ordinary education appear to be doing well, despite the disadvantage of having spent most of their formative years in a hospital with imbeciles and idiots.

The length of the children's stay in hospital ranges from 3 months to 9 years 2 months, the average stay being 4 years and $2\frac{1}{2}$ months. Five children remained for less than a year. Three of these were of average intelligence and are to be found in Table I under "Still at school". The other two had adverse social backgrounds, one had a triple handicap of partial sight, maladjustment and dull intelligence, and had been moved from his home by the Juvenile Court at a very early age. The other was brought up in a nursery having been removed at 2 weeks from his mother, who was a patient in a hospital for mental defectives.

There were five who remained in the hospital over 7 years prior to transfer to suitable schools. Two of these have failed in their trial at special schools, two have completed their education and are now at work, and another is doing very well at a normal school for the deaf.

The average length of trial given to the nine who have been excluded is approximately 2 years. The shortest was 6 months, and the longest trial was 3 years.

Of the children still at school there is one boy who spent six years at this hospital before being transferred to a school for the deaf. He won a scholarship to a technical school for the deaf and at present his aim is to become a veterinary surgeon. On admission to this hospital his behaviour was very disorganized and the conflicting verdicts on his deafness made it difficult to secure his acceptance as an educable deaf child. The fact that eighteen of the thirty children still at school have "additional handicaps", shows the difficulty and importance of accurate assessment.

Some of the children were given a trial at a local school before being accepted as educable, also a number were transferred "on licence" to residential schools. One child who failed while on licence at a residential school for children with deafness and additional handicap was excluded from school. He has now been placed in a hostel and is attending a normal school for the deaf. His faulty habits and sense of insecurity were the cause of his exclusion from his first school. His behaviour is not surprising considering he had spent 7 years 2 months in a hospital with low-grade children. Such a change of routine and environment demands a great deal of a child and is particularly difficult for the deaf child.

One child of 3 years was admitted as a matter of urgency. His behaviour at home had been extremely disturbed, destructive and tended to disrupt the family. The home had to be organized to prevent the child causing too much material damage. He caused sleepless nights and exhausting days. After a period in hospital in which the disturbed behaviour subsided, it became evident that the child was educable. He was given a trial at an Educationally Sub-normal Day School; accepted as educable and is now at home. His behaviour and educational attainments are satisfactory.

III. THE NORMAL CHILDREN

The circumstances of the three children found to be of average intelligence on admission are shown in Table II.

TABLE II

The Circumstances of the Three Children Found to be of Average Intelligence on Admission

Child	Age at 1st Ascertainment		Age on Admission		Length in Fountain Hospital (Months)	Relevant Home Circumstances Before Admission	Age at 1 July, 1956		Discharged to:	Circumstances on 1 July, 1956
	Y.	M.	Y.	M.			Y.	M.		
A ..	1	6	3		5	1. Illegitimate. 2. Mother in the care of Council.	7	2	Residential Nursery.	1. Boarded out with foster parents. 2. Attending Primary School.
B ..	1	3	2	7	8	1. Parents separated. 2. Child rejected. 3. Kept in General Hospital 1 year awaiting admission to Fountain Hospital.	8	4	C/o Father, Attending Day Nursery.	1. Boarded out with foster parents. 2. Attending Primary School.
C ..	2	5	4	5	3	1. Large family. 2. Child rejected. 3. Very dirty habits, evidence of emotional disturbance.	4	6	C/o foster parents.	1. Residential home. 2. Being examined for ? E.S.N.

Child "A" was described as "the product of folk of poor morals". His mother was illegitimate and was in the care of the Council, he also had the disadvantage of illegitimacy. The admission certificates portray a child who is retarded in all aspects of his development—especially in speech. The ordeal for a small child, aroused early in the morning to spend several hours awaiting two medical examinations must be severe, and it is not surprising that a record may contain a comment that the child "gazes vacantly at times".

This child was first ascertained as mentally defective at 18 months and was certified on admission at 3 years. Shortly after admission the psychologist found he had an I.Q. of 95 on the Merrill-Palmer test if the speech items were eliminated. Her report states: "He was at first silent, but started to talk."

Four years later this child is attending a normal primary school, his speech is normal and he is happy with his foster parents.

Child "B" was referred to the local authority at 15 months by a children's hospital. There was considerable marital disharmony at home and when the mother became pregnant for the third time this child was admitted to a residential nursery. Owing to infection he was transferred to a general hospital and remained there for twelve months. He was not ill but the mother felt unable to manage him as well as the two normal children.

On admission it was found that this child's intelligence level placed him in the "dull and backward" group. His behaviour showed signs of insecurity. After a period in the care of his father and daily attendance at the local Day Nursery he improved greatly. When taken into care by the children's officer he was observed in residential care and the psychiatrist's report stated: "I formed the opinion that he is not mentally defective." It was also recorded that the child was certainly backward in his development and that "contributory factors" are his transfer to a nursery and then to a hospital and later to the Fountain Hospital. These three moves and separation from his mother, added to his already unfavourable experience, are bound to have a serious effect.

It is encouraging to know that this child is now doing well with foster parents. He attends a Primary School.

Child "C" was referred to the local authority for ascertainment by the Child Guidance Clinic at the age of 2 years 6 months. He was not considered certifiable and a further examination was undertaken a year later. It was noted that he did not respond to any tests at all and said "no" to everything.

He was admitted to this hospital at 4 years 6 months. The home was poor, the father attending a psychiatric clinic and frequently out of work. The large family was anxious for the "defective" child to be removed as soon as possible. His habits were very dirty and he was a nuisance at home. The local authority arranged his transfer to foster care under the children's officer. In July, 1956 it was reported that this child was likely to be transferred to a residential school for the Educationally Subnormal.

IV. THE CHILDREN EXCLUDED

Table III shows some factors in the social background of the nine children who have failed in their trial.

TABLE III

The Nine Children Subsequently Excluded as Ineducable under Section 57(3) of the Education Act

	Age at 1 July, 1956		Length of Time in Fountain Hospital		Illegiti- mate	Relevant Factors in Parents	Pre- vious Institu- tions	Age on Transfer		Approx- imate Period at School	Circumstances at 1 July, 1956	
	Y.	M.	Y.	M.				Y.	M.	Y.		M.
D ..	9	10	4	10	+	M—psychotic	6	6	8	9	M.D. Hospital.	
E ..	8	4		6	+	M—M.D.	1	3	9	3	0	M.D. Hospital.
F ..	15	5	6	0	+	M—psychotic	3	11	5	2	0	M.D. Hospital.
G ..	12	1	1	1	+	M—abandoned child	5	6	0	3	0	M.D. Hospital.
H ..	20	9	1	1	—	Nil	1	13	9	2	0	M.D. Hospital.
I ..	12	3		6	—	M—deaf	—	4	3		6	M.D. Hospital.
J ..	15	8	6	1	—	F—psychotic	—	12	6	3	0	At home.*
K ..	16	10	9	2	—	Nil	—	13	0	2	0	At home.*
L ..	16	11	8	11	+	Nil	4	12	7	2	0	In Hostel at work.

* Attending Occupation Centre. M=Mother F=Father

Five of the children excluded were illegitimate compared with 11 in the total of 42. Illegitimacy is a social disadvantage and all five were separated from their mothers at an early age: in addition they had several placements before being admitted to this hospital. One illegitimate child who was excluded from an Educationally Subnormal School after a period of 2 years was fortunate to avoid admission to a Mental Deficiency hospital by being placed under Guardianship. He is living in a hostel provided by a voluntary society and is working in a local factory.

Three children have a parent who is a chronic patient in a mental hospital. The mother of one child is in a Mental Deficiency hospital. Another child was found to be psychotic and not deaf. He failed at the School for the Deaf and was admitted to a Mental Deficiency hospital. He has also had two periods of treatment in a mental hospital. One child was found abandoned. This makes a total of 7 of the 9 excluded children who were handicapped by adverse social circumstances.

The age of transfer seems relevant. In Table III it is shown that five of the nine children were between 11 years and 14 years of age at transfer. However, amongst the children who have completed their education there are three who were transferred after 12 years; nevertheless transfer at an earlier age seems advisable.

In a large children's hospital where social stimulation is limited, the child is often denied many normal social experiences, some of which are necessary to a satisfactory adjustment to community life. With the help of the Parents' Association it has been possible to give a few children the advantage of a regular visit from an "Aunt" or "Uncle". This has proved valuable and the children have benefited greatly.

V. THE CHILDREN WHO HAVE COMPLETED THEIR EDUCATION TO 16+

The circumstances of the five children who have completed their education is seen in Table IV.

TABLE IV

The Circumstances at 1 July, 1956 of the Five Children Who have Completed Their Education to 16+

	Age at 1 July, 1956		Period in Fountain Hospital		Illegiti- mate	Institu- tions Before Admis- sion	Relevant Factors in Family	Approximate Period at School		Circumstances at 1 July, 1956
	Y.	M.	Y.	M.				Y.	M.	
M ..	19	1	5	9	—	None	Mother dead.	4	—	Died 1955.
N ..	16	9	8	4	—	3	Mother M.D. and psychotic	5	3	Was at home and at work.
O ..	16	10	5	4	+	2	—	6	6	M.D. Hospital.
P ..	16	11	8	0	—	—	—	5	—	M.D. Hospital.
Q ..	16	11	5	1	—	—	Father psychotic	7	6	At home and at work.

It was encouraging that one of our boys chose to visit us very shortly after commencing residential domestic work. His father had died and his mother could not be traced. She was mentally defective and had had several periods of treatment in a mental hospital. All her children were in the care of the local authority. On leaving school this boy was re-certified and admitted to a Mental Deficiency hospital. He was placed out in work within a few months.

The extent of hostel provision for educationally subnormal school leavers is very limited. A boy who is capable of completing his education is likely to benefit more from hostel life than from admission to a Mental Deficiency hospital. The educationally subnormal school leaver without a suitable home has a great handicap. Certification in adult life is a great social disadvantage. The fact that a boy was suitable for certification under the Mental Deficiency Act tends to be written in his history and produced at every social crisis. The interpretation that society may not be providing suitable accommodation is rarely considered. Certification remains a social disgrace. Every child completing his education should be given the opportunity of showing his competence to remain in society.

VI. PSYCHOLOGICAL ASPECTS

Since the children varied widely in age on transfer, and some had motor or sensory handicaps which necessitated a variety of tests, the intelligence quotients (I.Q.s) are not comparable; consequently a comparison is not justified between the children excluded from or retained at school. The 41 I.Q.s are, however, given in Table V in order to indicate the wide range of ability, 12 scoring 70 or above. It will be noted that the excluded children tend to fall in the lower ranges.

Examination of the cases in this survey reveals that conditions other than the low I.Q. are more likely causes of educational failure. The previous analysis (Table III) has shown that 7 of the 9 children who were excluded were either transferred at a late age (over 11 years) or had had the disturbing experience of numerous placements in their early years, in 2 cases both factors being operative.

The educational problems of transfer to school at the age of 11 and 12 years need no further discussion.

Subsequent emotional disturbance resulting from the frequent changes of homes could interfere with school learning. Consequently information was sought on the response of these 42 children to problems in an intelligence

TABLE V
Last Test Result Before Transfer

I.Q. Range					Children Still at School or Left at 16	Children Excluded	Totals
					N	N	N
100-109	..	..	..	..	1	0	1
90- 99	..	..	..	..	1	0	1
80- 89	..	..	..	..	1	1	2
70- 79	..	..	..	..	8	0	8
60- 69	..	..	..	..	11	2	13
50- 59	..	..	..	..	9	6	15
40- 49	..	..	..	..	1	0	1
Not tested	..	..	..	..	1	0	1
					—	—	—
					33	9	42

test situation and on their ability to apply themselves to simple tasks at the Occupation Centre. Reports were examined for references to such behaviour as distractibility, persistence, lack of it, ability to concentrate, etc.

Reports were not available for 2 children: one was the child who was excluded from a school for the deaf and subsequently found to be psychotic; the other was a normal child who is now attending a primary school.

In 16 cases there was either a positive reference to persistence or ability to concentrate, or absence of a remark on distractibility, etc., from which it may be concluded that there was nothing in the child's behaviour to cause comment either way. Of these 16 cases only 2 were excluded from school, and one of them is the boy who subsequently obtained and held employment (Binet I.Q. 55 at the time of transfer). The other is reported by the psychologist of the mental deficiency hospital to which he eventually went as having a Binet I.Q. of 28, and he thus appears to have deteriorated intellectually (Merrill-Palmer I.Q. 51 on transfer).

Of 8 children whose early reports of behaviour in the test situation or Occupation Centre noted distractibility, etc., but whose later reports remarked on improvement in these respects, only one has been excluded from school. She had lived in an institution most of her life, and was 12½ years old when transferred.

Four children who were distractible when being tested, but who were reported to concentrate well in the Occupation Centre were all retained in the educational system.

Nine children consistently manifested distractibility or lack of persistence in the test situation, and in the Occupation Centre. Another was noted in later reports as showing this behaviour, but not in earlier ones. Of these 10 children, 5 were excluded from school and another is reported to be making poor progress.

The results of this analysis are summarized in Table VI. The children who had improved or who could concentrate at the Occupation Centre but not in the test situation, were included in the not distractible group. Numbers are rather small for any definite conclusion; nevertheless the proportion of distractible children excluded is much higher than among those who were able to concentrate in a school situation at the time of transfer. If χ^2 (with Yates' correction) is applied to these figures, p is almost at the .02 level for 1 d.f. ($\chi^2=5.2$).

TABLE VI

Behaviour in the Occupation Centre and Test Situation of Children Retained in or Excluded from School

	Retained	Excluded	Totals
Distractible	5	5	10
Not distractible at time of transfer	27	3	30
	32	8	40

It is interesting that 5 of the most distractible children were retained at school: four of these, however, came to the Fountain Hospital straight from home, and the fifth had had only one previous change. Moreover, four were in regular contact with their parents while at school, and the fifth was visited regularly by a member of the Parents' Association which arranges such visiting. Four of the five excluded and distractible children, on the other hand, had no homes and were visited by no one; although the fifth was regularly visited by her father, he was psychotic. In two of the five excluded cases the Fountain Hospital was the sixth and seventh placement respectively before the age of 5 years, and in two others it was the fourth or fifth. One of the latter also had the handicaps of deafness and a late age at transfer.

One excluded child was psychotic, and if distractibility with consequent learning difficulty is regarded as indicating emotional disturbance, then 7 of the 9 excluded children appear to have been disturbed (1 of the remaining 2 seemed to have deteriorated intellectually and the other was transferred at 12½ years). Thus in most cases emotional disturbance rather than inferior ability appears to have been responsible for learning difficulties and exclusion from the educational system, and it seems likely that the emotional problems resulted from the frequent early changes or care by a psychotic parent. If the problems could have been alleviated, these children would probably have made educational progress. The findings suggest that going home to stable parents, or having the consistent interest of another adult, gives distractible children sufficient security to enable them to concentrate to some extent on school work. That 9 children with I.Q.s between 50 and 59 remained at school, indicates that children of this level of intelligence, who have lived in an intellectually unstimulating environment for several years, can benefit from education in an E.S.N. school; it cannot therefore be concluded that such children in mental deficiency hospitals should not be transferred to the educational system. Since, however, schools for maladjusted children are reluctant to accept children of low intelligence, and since the E.S.N. schools are adapted for the stable child, there appears at present to be no provision for the maladjusted E.S.N. child, for the mental deficiency hospital is similarly not suited to their educational and therapeutic needs.

VII. CONCLUSIONS

The findings of this survey provide further evidence of the disturbing effect of long experience of insecurity in the child's early years, and they also suggest that a series of changes is most disturbing. The number of previous institutions listed in Table III is revealing, and those children who have had the misfortune of being illegitimate have the added disadvantage of several changes in environment. If the child is abandoned or has to leave home for some reason, it seems preferable to keep the subsequent changes to a minimum. Moreover for the E.S.N. child who fails in his trial at school, and where there

is no alternative provision, it would be an advantage if he could return to the original hospital and be spared the need to adjust to yet another environment.

The three children who were found to be of average intelligence on admission were reacting to their previous adverse environment. Frequently a disturbed child of normal intelligence may superficially present a picture of backwardness.

The provision of hostel accommodation for educationally subnormal children outside the Mental Deficiency Act, is not a new suggestion, but the need is shown in Table IV. Two boys, both with no home, were transferred to Mental Deficiency hospitals on completing their education. Fortunately they are now at work, but were they really needing "hospital care"?

VIII. SUMMARY

1. A follow-up survey is reported of 42 children who, after a period in a mental deficiency hospital, were accepted as educable by local authorities.

2. At the time of the follow-up, 33 were still at school or had left school at 16 years: 9 had been excluded from school.

3. The 3 normal children in the group had experienced parental rejection or separation before admission.

4. Six of the 9 children subsequently excluded from school had undergone severely disturbing experiences before admission.

5. The intelligence quotients of the total group of children ranged from 49 to 102.

6. The excluded children tended to be more distractible than those retained at school.

7. Some implications of the findings are discussed concerning the effects of frequent changes of institution, the ascertainment of mental defectives and the provision of hostels for E.S.N. school-leavers without homes.

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BLINK RATE AND MUSCLE TENSION

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THE present paper will deal with eye blink frequency and its relationship to scores on "personality" questionnaires and to frontalis and right forearm muscle tension measured electromyographically. This analysis forms part of a larger experiment with the main aim of investigating levels of muscle tension in four groups—(i) introverted neurotics, (ii) extraverted neurotics, (iii) extraverted normals and (iv) introverted normals—during relaxation and while responding to a series of questions about neurotic symptoms.

Two problems are involved. First, it is a common clinical impression that a high blink rate is present in states of anxiety. Meyer (12) suggests that blink rate is facilitated in anxious subjects by their characteristically widespread muscular activity. In fact, there is no direct experimental evidence on this point. Meyer, Bahrick and Fitts (13) obtained no significant correlation between frequency of blinking and the Taylor Manifest Anxiety Scale (using normal subjects).

Meyer (12) has also attempted to develop a rationale for the use of blink rate as an index of generalized, i.e. widespread muscle tension. This poses the second problem with which this paper is concerned, and requires some preliminary examination.

The rationale derives from his theory of "efferent neural interaction", described in terms of the convergence of impulse patterns upon the motor pathways of the central nervous system and offered to account for the effects of induced muscle tension upon learned and unlearned responses. Meyer discusses the results of several experiments to support his argument. The data he presents all concern blink rate changes during the performance of differing tasks—mostly, but not all, of a visual nature—and provides evidence only of an indirect kind since there are no detailed correlations of eye blink changes with other simultaneous measures of muscle tension. Not only generalization but magnitude of local muscle innervation he claims may produce variations in blink rate. Drawing on the cortical representation work of Penfield and Rasmussen (14) in this connection Meyer argues that the "blink response is facilitated by simultaneous activity in neighbouring motor channels and most important motor channels fall in this category".

Subsequently, Malmo and Smith (9) in a paper concerned with the inter-relationships of various tension and motor measures, have presented some findings with respect to blink rate and proximal muscle action potentials. Since the cortical representation for the forehead is adjacent to that for eyelid movement they argue, on the basis of Meyer's reasoning, that a correlation between level of forehead tension and frequency of blinking would be expected. No significant relationship was observed in their data. Additionally, they examined the muscle potential from the neck, the motor representation of which is nearly as close to the eyelid as is the forehead, but again with no significant results.

Acceptance of the validity of Malmo and Smith's data is difficult on several counts. First, as they themselves indicate, the measure of blink rate was based on artefacts (slow wave changes) occurring in primary tracings of frontalis muscle potentials; such changes "may also accompany eye movement and the like". The exact relationship of such slow wave artefacts to eye blink is not specified and appears to be unknown. In our experience with similar records, large, slow wave deflections are most frequently associated with eye movements, whether the eyelids are open or closed (although apparently more marked when open). Normal eyeblink is less clearly recorded, although we have found that exaggerated blinks, involving a larger degree of the surrounding musculature, do produce a noticeable wave change in the frontalis record.

Second, the ambiguous comment appears (p. 403) "nor, since our Ss had their eyes shut, is there any factor of visual control to complicate the findings". It is difficult to see how blink rate could be assessed under this condition. Third, it is not clear whether part, or whole, of the experimental data for forehead muscle potential was used. Shifts in relationships between tensional foci, and changes in the magnitudes and distribution of tensions as a result of differing experimental conditions (i.e. rest and stimulus) might reasonably be expected, and might be obscured by an overall correlation.

Malmo and Smith are not unaware of the shortcomings of their data, and themselves stress the need for careful simultaneous recordings of muscle potentials and blink rate in order to test Meyer's conclusion.

The present experiment was designed to record eyelid activity, and frontalis and right forearm muscle tension simultaneously. Thus some further check on the degree of correlation between these measures could be made. Specifically, the two problems with which the present paper deals are:

(i) Does the eyeblink rate differ in groups selected on the basis of neuroticism and introversion-extraversion, either during rest or during a period of questioning?

(ii) What relationship exists, under these two conditions, between blink rate and forehead and arm muscle action potentials recorded simultaneously?

METHOD

SUBJECTS

Four groups of male university students were selected by means of a neuroticism and an introversion-extraversion questionnaire (the dimensions of neuroticism and introversion being orthogonal). Details of these scales and their derivation have been given by Eysenck (6) and Franks (7). Only extreme cases from the four quadrants—neurotic introverts, neurotic extraverts, normal introverts and normal extraverts—were tested, and there was no overlap in the scores on neuroticism and introversion-extraversion. According to Eysenck's theory (5) the hysteric forms the prototype of the extraverted neurotic, the anxiety state of the introverted neurotic.

PROCEDURE AND APPARATUS

The questions asked of the subjects during the questioning period were derived from a neuroticism rating scale described in *The Dimensions of Personality* (4). There were 11 questions, all concerned with neurotic symptoms (sleeplessness, irritability, etc.) presented at a fairly uniform speed of one per 20 seconds. The subjects were instructed to give brief replies concerning the presence of such symptoms in themselves. Earlier results had shown a very

significantly larger increment of forearm tension in neurotics than in normals during this situation (11).

The outline of the complete experimental procedure is as follows:

- (a) Initial rest period—3 minutes—eyes closed.
- * (b) Rest—2 minutes—eyes open.
- (c) Rest—2 minutes—eyes closed.
- (d) Instructions—1 minute—eyes closed.
- * (e) Questioning—3 minutes 20 seconds—eyes open.
- * (f) Rest—2 minutes—eyes open.

The present analysis deals only with those periods marked by an asterisk.

Simultaneous recordings were obtained from three sources: the frontalis muscles, right forearm extensor, and left eyelid.

For eyelid recording, two EEG cup-type electrodes (1 cm. diameter) were placed adjacent to the outer orbit of the left eye in a vertical position, separated by approximately $2\frac{1}{2}$ cm. Collodion was used to fix the electrodes. To avoid eye-watering from the collodion vapour, electrodes were always attached while the eyes were closed, and Ss were instructed not to open their eyes until the collodion had set firmly. A period of adjustment was allowed before recording commenced.

Three single-channel Ediswan electro-physiological amplifiers were employed; the primary tracing was recorded on an inkwriting pen oscillograph unit with a paper speed of 1.5 cm. per second. Muscle activity from the forehead and arm was scored from a continuous integrated record which responded mainly to amplitude changes. Eye blinks were counted from the primary tracing. A typical record obtained of blink activity is given in Figure 1.

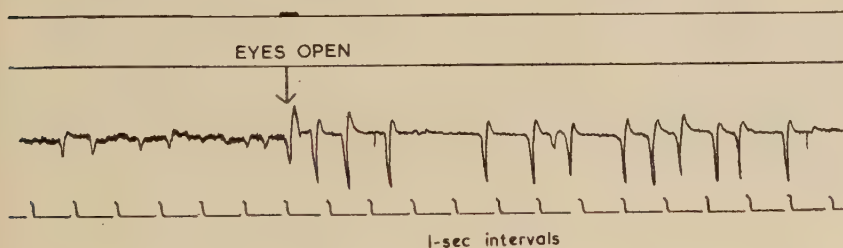


FIG. 1.—Eyeblink tracing: electromyographic recording.

On most occasions individuals presented a clearly defined blink pattern, but variations in amplitude and shape did occur, and the decision to score a blink was not always unequivocal. An amplitude was selected by inspection of the raw record below which no wave was counted; as far as possible this amplitude was held standard throughout the scoring. Total number of blinks occurring each 10-second period was scored.

Deflections from the base-line record could also occur as a result of rather large eyeball movements. The subjects were, however, lying relaxed on a hospital bed, facing a plain ceiling, and were requested very carefully not to make any movements, particularly of the head or arm. No visual fixation point was provided. A considerable amount of preliminary attention was given to the shape of the curves produced by eyeball movements, and it is not considered that these influenced the measurements obtained in the present experiment.

FACTORS OF AGE, TIME OF TESTING, VISUAL CORRECTION AND LIGHT INTENSITY

- Age
- Group 1—Introverted Neurotics. N=15; average age 23·8 years.
 - Group 2—Extraverted Neurotics. N=17; average age 22·3 years.
 - Group 3—Extraverted Normals. N=17; average age 22·5 years.
 - Group 4—Introverted Normals. N=18; average age 23·7 years.

The age range was quite small, and this factor did not affect any of the measurements.

Time of Testing

Half of each group was tested in the morning (9.0 a.m.) and the other half in the afternoon (2.0 p.m.). There is a tendency for a higher blink rate to occur in the afternoon period, probably related to different a.m. and p.m. illumination intensities (discussed further below).

Visual Correction

Those subjects wearing glasses were requested to remove them both because of the nature of the electrode attachments and the lying position necessary during the experiment. These subjects comprised 6 from Group 1, 6 from Group 2, 5 from Group 3, and 5 from Group 4. Their blink rate was compared with that of other subjects; a slight but not significant increase was observed.

Light Intensity

Testing was almost always carried out in normal daylight, but on a few occasions artificial illumination was used. This light, however, was placed behind the subject's head; there was no direct glare over his eyes. A reading of the light intensity was taken at the end of the experiment by means of a light meter placed in the position of the subject's eyes. The range of foot-candles readings was quite small (2–19) and within this narrow range no influence on blink rate would have been expected. In fact, a negative correlation (-0.375) was obtained between the light intensity and frequency of eyeblink. This ($N=44$) is significant at the 1 per cent. level.

RESULTS

Blink Rate and the Personality Dimensions of Neuroticism and Introversion/Extraversion

The means of eyeblink frequency for the four groups are given in Table I; the analysis of variance table obtained from these data is given in Table II.

TABLE I
Rate of Blinking

(Average number of blinks during each period are given. For purposes of comparison all periods are reduced to 110 seconds.)

	Group 1 Introverted Neurotics N=15	Group 2 Extraverted Neurotics N=17	Group 3 Extraverted Normals N=17	Group 4 Introverted Normals N=18
Introductory rest ..	33·80	34·35	31·82	30·72
Questioning ..	64·35	66·82	59·00	61·78
Final rest ..	43·13	37·06	40·53	30·89

TABLE II
Analysis of Variance: Eyeblink Data

	df	msv.	F	
Main Effects:				
Introvert/Extravert classification	1	125·008	4·976	N.S.
Normal/Neurotic classification ..	1	736·098	·845	N.S.
Conditions	2	15548·376	24·995	Significant beyond ·0001 level.
1st order Interactions:				
I/E with No./Ne.	1	82·680	7·524	N.S.
I/E with conditions	2	42·917	14·494	N.S.
No./Ne. with conditions	2	27·034	23·001	Significant 5 per cent. level.
2nd order Interactions:				
I/E with No./Ne. within conditions	2	550·605	1·202	N.S.
Residual variance between persons of the same classification in a given condition				
	168	622·062		
Total Variance: 137788·1	179			

The F ratio based on "conditions" is highly significant, indicating that a very large part of the observed variation can be attributed to the effect of changing experimental conditions, in this case from rest to questioning.

The fact that there is such a highly significant increase in blink rate during questioning over resting level is of interest in demonstrating the important effect on eyeblink of the act of speaking. Further testing might indicate whether this large increase was due to speaking *per se* or to the "stressful" effects induced by the specific questions used.

Although the means of the neurotic groups tend to be somewhat higher, variation in blink rate is large, and no significant effects from the classification into normal/neurotic, or introvert/extravert, were found. (A significant interaction of the normal/neurotic classification with conditions was obtained; this ratio in fact reflects a significant concordance, not difference, between groups, in so far as the variation observed between groups within conditions is less than would have been expected by chance.)

It will be recalled that a significant correlation was obtained between blink rate and light intensity. As a result of this, the group means for light intensity were calculated. Unfortunately light meter readings were taken on only a limited number of occasions (N=44), fairly equally distributed, however, over the four groups. The mean light intensities given in Table III were therefore

TABLE III
Illumination Levels During Testing
(N=44)

	Int. Ne.	Ext. Ne.	Int. No.	Ext. No.
Foot-candles	6·2	3·8	6·6	7·2

derived from small numbers; it can be seen that in three sub-groups the room lighting appears to have been fairly similar—in one, the extraverted neurotics, a rather lower level of lighting was obtained.

The possibility must be considered that the lower illumination during testing of Group 2 contributed to a slightly increased blink rate, and it is regretted that the results on group differences presented in this section must be regarded as somewhat inconclusive. Re-test of these findings is obviously required. It is felt, however, that the available evidence does not appear to support the impression of higher blink rate in neurotic subjects.

Blink Rate and Other Tension Measures

Product-moment correlations obtained from measures during the initial resting period were as follows:

Eyeblink and forehead: $+0.022$ ($N=71$) N.S.

Eyeblink and arm: -0.013 ($N=72$) N.S.

No relationship was observed between frontalis or arm muscle action potential and rate of blinking during rest.

Correlations obtained from questioning period data were:

Eyeblink and forehead: $+0.368$ ($N=68$) Significance 1 per cent. level.

Eyeblink and arm: $+0.140$ ($N=70$) N.S.

The significant correlation of $.368$ points to the tendency for high blink rate to be associated with a high level of forehead activity during questioning.

Another feature of the data was investigated: this was the consistency of eyeblink frequency from one period to another. The initial and final resting session eyeblink rate correlated to the extent of $+0.616$. Initial rest and questioning period measurements correlated $+0.488$. Both coefficients are, of course, quite significant ($N=72$).

A graph showing the trends apparent in eyeblink frequency during the experiment is reproduced in Figure 2.

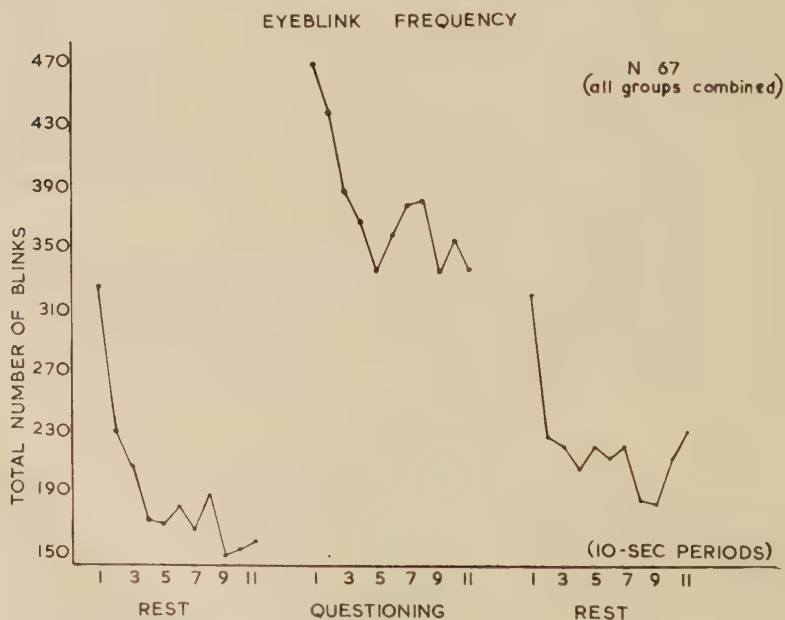


FIG. 2.—Frequency of eyeblink during rest and questioning.

* In each case the correlation coefficient was calculated on the largest N possible; incomplete records have reduced the N in several of the correlations.

DISCUSSION

The analysis of variance showed no significant effects arising from the classification into normal/neurotic, or introvert/extravert. Two additional findings were: (i) a very large variation in respect of rate of blinking, and (ii) an unexpected correlation between blink rate and illumination level. It is not considered, however, that the present results support the widely held impression that blink rate is significantly higher than normal in neurotics, or introverted neurotic (i.e. anxious, obsessional) subjects.

It is recognized, of course, that "neurotic" or "anxious" subjects selected on the basis of questionnaire scores may differ in certain important respects from the hospitalized groups, and it is hoped to repeat this experiment using psychiatric diagnosis as a criterion.

During the questioning, all subjects' blink rates were very significantly increased. It would be interesting to investigate this aspect of the data more fully since, on the available evidence, it is not possible to differentiate the effects of speaking from the more complex effects of "stressful" questioning.

In terms of Meyer's predictions one of the more interesting features of the data is the observed correlation between forehead tension and eyeblink rate during questioning, but not during rest. In assessing the relevance of this finding to his discussion, several factors, possibly interacting, should be considered.

(a) The type of situation in which the correlation is found (the act of verbally responding to questions) almost certainly necessitates an additional and *widespread* degree of activity, particularly of the facial musculature. Yet in the present experiment a highly significant innervation of arm musculature was observed in all groups ($t=8.65$, $p<.0001$) which was uncorrelated with either forehead tension or eyeblink rate. This suggests quite a complex distribution of muscular tensions within a widespread response.

(b) The distribution of tension during rest and stimulation probably varies widely from individual to individual; this may result in a given level of frontalis tension producing different degrees of facilitation. In any case, distribution and magnitude of tensions being equal, it seems possible that threshold excitability may differ from individual to individual.

(c) Temporal factors are also of great importance. Any investigation, even of so-called simultaneous responses, would be incomplete without reference to the nature of the separate changes (habituation, for example) which occur as a function of time alone. It is difficult, *a priori*, to select those time intervals which may fairly reveal a relationship between two rapidly changing functions. The present use of a statistical correlation based on two measures obtained over a period of minutes may well be questioned.

As a check on this latter point, the data were examined with respect to trends which might occur during the time periods for which measures were obtained. The ten-second readings for eyeblink rate are plotted in Figure 2. Initially large downward slopes are apparent. Frontalis tension, on the other hand, remains almost steady within the separate periods, and manifests no observable trends. It seems likely that the degree of correlation existing between the two measures varies considerably as a function of the time intervals selected. Why this should be is not clear from Meyer's theory.

It would seem to be necessary, then, in evaluating tensional effects on eyeblink, first to ascertain the total tensional pattern occurring during the questioning condition; this would provide exact quantitative data on the extent

and magnitude of muscle innervation. The course of changes along the time axis must be taken into account (i.e. adaptation, latency) and individual differences in the total response pattern considered. A more precise assessment of the relationship between individual muscle functions could, on this basis, be the more readily made.

The total electromyographic pattern occurring during speech has not been ascertained, but Smith, Malmo and Shagass (16) have found significant increases over resting tension not only in chin (speech) muscles, but in right arm, left arm, forehead and neck muscles. The authors were not primarily interested in the interrelationships of these measures, and so do not give a correlation matrix of their data. It cannot be assumed, however, that these tensional increases are intercorrelated: in another context (9) the results of a factorial analysis indicated poor correlation of frontalis tension with that of five other muscle groups. Since the important cortical projections for the face and hand are relatively close, the lack of relationship is surprising, especially in view of Mayer's comments, although in agreement with present findings.

Although Meyer has predicted that induced muscular tensions should have an effect upon both the blink rate and the blink reflex, "by making the motor system available to an input, and not by operating on the input", experiments relating reflex to induced tension, which he quotes, reach no firm conclusion. His argument seems to leave out of account many of the findings arising from electromyographic studies, not the least important of which is the generally low intercorrelations observed even between adjacent muscle measures. As Davis (3) has observed "no one point in the musculature could be relied upon to show the state of tension in the whole body". Meyer's conclusion that "one criterion of anxiety is an unusual amount of muscular activity" therefore requires qualification concerning location, magnitude, and conditions under which the tensional phenomena are present or absent.

If one accepts that only detailed electromyographic evidence can provide information about the distribution of muscle tensions then it must be admitted that Meyer's arguments are based on experiments employing largely indirect or inadequate means of quantifying tensional states. Precise measurement of the magnitude and distribution of muscular tensions during the performance being studied is needed in order to determine more exactly (*a*) the facilitating or inhibiting effect of muscular tension upon performance, (*b*) the nature of changes occurring in time, (*c*) the efficacy of blink rate changes as indicators of overall tension changes, (*d*) the extent to which blink rate is affected by the magnitude of adjacent muscle innervation, and, of course, the interaction between (*c*) and (*d*).

The large increase in arm tension, and relatively smaller although significant increment in forehead tension ($t=5.245$ between rest and questioning) found in the present experiment, yet the correlation with eyeblink of the latter, rather than the former, are facts whose prediction is not clear from Meyer's hypothesis. The need to take into account differential trends occurring during quite short periods of time has not, perhaps, been sufficiently stressed, especially since facilitation of motor responses could presumably occur with great rapidity.

In the absence of detailed information concerning muscle tensions during specific conditions, changes taking place from rest to stimulus, and within periods of rest and stimulus, it is impossible to gauge the effectiveness of eyeblink frequency as an indicator either of overall, widespread tension, or as a facilitated response occurring from its interaction with proximal (frontalis) tensional levels.

Although, as argued by Meyer, the use of blink rate obviates the employment of elaborate instrumentation to detect changes in the distribution of muscular tension, it is important to remember that the available literature testifies to the fact that eyeblink is readily altered by a large number of extraneous influences. The ease of measurement may, therefore, be outweighed by the number of experimental controls which need to be exercised.

Psychological reasons apart, there are innumerable physiological conditions of the eye which might affect blink reflex and blink rate; Luckiesh and Moss (8) have listed some of the relevant variables—intensity of illumination, duration of task, size of visual material, presence and absence of glare, and refractive errors.

The trends apparent in the eyeblink data suggest that changes in rate occur within short periods of time, both when the subject is unoccupied and when he is responding to questions. The first eyes-open resting session measurement followed a 3-minute recording period during which the eyes were closed, and it is possible that the immediate high rate upon opening the eyes is incurred during the process of light adaptation. Similarly the questioning period followed upon rest with eyes closed, although the immediate response on this occasion is very much greater. The final rest period, however, followed immediately upon the cessation of the questioning, during which the eyes had been open.

Meyer reviews a large number of psychological experiments in which eyeblink changes were recorded. Reading, working arithmetic problems, various clerical tasks, distant and near fixation, time and effort expended upon a visual task, mirror drawing and emotional disturbances have all been shown to affect blink rate, some decreasing, others increasing the intra-blink period.

The present findings of increased blink rate attributable to illumination intensity, and to a slight extent by impaired vision, point to the need for careful controls, particularly as small errors may well summate in a particular direction. It is interesting to note that Luckiesh and Moss (8) also found increasing blink rate under a lower level of illumination (using intensities of 5, 10 and 100 F.C.) but that this finding was confined to the last five minutes of an hour's reading. Initial differences were not found. Meyer draws attention to several studies which fail to confirm the relationship of blink rate to illumination, and refers to a study by Ponder and Kennedy (15) in which blink rate was found to be unaltered over a wide range of light intensities and even in total darkness. Some settlement of this outstanding question would be of great value.

Another factor to be recognized is the high variability of blink rate "not only from subject to subject and from one occupation to another but from minute to minute in any one subject engaged in a set task" (1). A high variability was also noted in the present data.

The reliability coefficient obtained of .616 ($N=72$) in the present experiment was derived from two short periods separated by a relatively brief time interval, and thus provides only limited information on the consistency of the measure. Follow-up studies taken over longer periods should provide useful basic information on this point.

SUMMARY

1. Simultaneous recordings of left eyelid activity, and frontalis and forearm muscle tension were obtained in four groups selected by questionnaires of neuroticism and introversion-extraversion.

2. The results did not indicate any significant differences in tension or blink rate between groups. Frontalis and forearm tension, as well as blink rate, increased significantly in all groups from rest to questioning.

3. A significant correlation between forehead tension and blink rate was found during questioning. Correlations between forehead and arm tension, and blink rate and arm tension, were not significant either during rest or stimulus conditions. The relationship of these results to Meyer's suggestion that blink rate could be used as an index of generalized muscle tension was considered.

4. It was not felt that the experiment provided a very adequate test of his hypothesis, and that additional detailed electromyographic information concerning the distribution and magnitude of muscle tensions during the experimental conditions would be necessary. The numerous factors influencing blink rate, the high variability of the measure, and the difficulty of validating it as a precise measure of generalized tension were discussed.

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PSYCHIATRIC SYNDROMES ASSOCIATED WITH CEREBROVASCULAR DISORDERS IN THE AFRICAN

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THIS contribution towards the subject is limited to three of its aspects—namely, the possible aetiological role of inherited predisposition and psychogenic factors (mainly environmental to which the patient is still exposed), the relationship between late endogenous depression and the symptoms of cerebrovascular changes, and prognosis. On the face of it, prognosis of course must ultimately depend on that of the cerebrovascular disorder but apparently hereditary and environmental factors play a more significant role. Inherited predisposition is here assessed in terms of constitutional and personality factors.

Ten patients suffering from hypertension with cerebral involvement and twelve from cerebral arteriosclerosis were carefully selected from a series of over sixty patients over the age of forty and studied within a period of two years during their entire stay in the neighbouring villages at Aro. It was observed that a surprisingly high number of patients manifested psychiatric disturbances which closely simulated the classical functional syndromes of psychoneurosis and affective disorders. In over 60 per cent. of the cases, the setting of affective disorders was indistinguishable from that of endogenous or involutional depression.

The subject of psychoses associated with hypertension and arteriosclerosis has been exhaustively dealt with by a few important writers (Mayer-Gross, 1937; Guttman, 1937; Rothschild, 1945, 1947a, b; Mayer-Gross *et al.*, 1954; Martin Roth, 1955), but Guttman (1937) has observed that very little attention has been paid to the group of vasolabile hypertension from the psychological standpoint.

Some of the contributions of the previous authors were supplements to the basic observations made by Krapf (1936), quoted by both Mayer-Gross and Guttman. Nevertheless, in spite of their classical contributions, it is well from time to time to inspect foundations among the dwelling-places of neuro-psychiatric theory; else by constant usage we are apt to gain from the appearance of the superstructure a false sense of the solidity of the whole. However, no less than a psychosomatic approach can throw adequate light on some of the anomalies and lack of correlation between the physical findings and the psychiatric symptomatology encountered in this field.

And yet, in spite of the uniqueness of our clinical material in that it is confined to a racial group that had hitherto been medically unexplored and of presumably different constitutional structure, no originality whatever is claimed for the views expressed here, since they have been advanced many times before by these competent authors, but the support they received from the cases examined in detail here is sufficient to place their views beyond the sphere of mere unverified hypothesis.

There has been little work done on the quantitative and qualitative aspects of essential hypertension among the Negro race as a whole, especially among the

indigenous population of Africa. At this stage, however, it is desirable to deal with the question of the alleged rarity of essential hypertension in indigenous Africans. Carothers (1951) has said that in many areas, e.g. South Africa, Rhodesia, and Uganda, hypertension and hypertensive heart disease are quite common. Carothers, however, went on to say that in other parts of tropical Africa its chief causes are nephritis and renal obstruction, and that essential hypertension is rare.

It is immaterial for the purpose of this paper what is the true position with regard to the occurrence of essential hypertension in the indigenous population of tropical Africa, but from a consideration of the clinical material, personal and otherwise, at our disposal, it does seem that essential hypertension is not uncommon among Africans living in rural areas, despite the fact that multiple aetiological factors for the manifestation of high blood pressure among native Africans must be accepted.

It is not unusual in tropical Africa to find that by the time patients seek treatment there has been varying degree of irreversible vascular damage, renal and cardiac pathology, secondary to essential hypertension.

Nothing definite is known about the incidence of arteriosclerosis in tropical Africa, although Carothers (1951) stated that it was common among the Southern Bantu. Lewis (1942), quoting Schwab and Schulze (1931), said that these authors working in a clinic in America found 42·4 per cent. of the white cases and 63·2 per cent. of the coloured cases were hypertensive, and 35·9 per cent. of the white and 13·7 per cent. of the coloured were arteriosclerotic. They concluded, however, that the low incidence of arteriosclerosis in Negroes might be a reflection of the lower proportion of the upper-age groups in that race.

With regard to the Western Region of Nigeria, since there are comparatively few old people among our population the question of the incidence of senile and arteriosclerotic psychoses must remain open. There is no doubt that a small proportion of them are manageable in their homes in towns and villages and therefore do not come under observation. Nevertheless, a great majority of them are found in various Nigerian Asylums, having been criminally charged and committed for wandering.

However, because of the psychiatric features, we see a comparatively higher proportion of arteriosclerotic patients than are normally seen in the ordinary medical out-patient. On the other hand, the lower proportion of the upper age-group in this race should not make a great deal of difference, since it is the impression of the author that organic psychiatric conditions that are usually associated with senescence in European and American countries occur much earlier* in the indigenous population of Nigeria.

It can safely be assumed that there are differences in the rate and mode of ageing (including cerebral degenerative diseases) between races† on which inherited predisposition may throw some light.

Moreover, Schwab (1935), quoted by Lewis (1942), on the results of his vasomotor tests based on those of Hines and Brown (1933), found that the

* Most of the hypertensive patients were over 40 years and the arteriosclerotic in the mid-forties. Arteriosclerotic patients in higher age-groups (i.e. over 60 years) seem to demonstrate this overlap or intrinsic relationships as forcibly as the age-groups of our present material, so that age in itself is of no prognostic significance. Dementia is, however, much more pronounced in the higher age-groups and this tends to confuse the picture.

† *Vide* Brown *et al.* (1948) on "Some remarks on premature ageing in the Eskimos", *Rev. of Canad. Biol.*, 7, 178 (1948). Also the preponderance of cerebrovascular disorders among the Japanese.

elevation of blood pressure was more marked in Negroes and concluded that "there is a quantitative racial difference in reaction to a standard vasomotor stimulus, indicating a more sensitive vasomotor mechanism in the Negro".

Racial or constitutional predisposition to vasomotor sensitivity, of which cerebral circulatory disturbance may be an important component, would seem to be associated with, or causally related to, early senescence. Thus the relationship of endogenous affective disorders to the psychiatric syndromes associated with early occurring organic cerebral disorder in this racial group is one of great practical interest. Therefore, the overlap between the two conditions is not only theoretically possible but found in clinical practice among Africans to be much greater than was anticipated. Moreover, in spite of the fact that the incidence of cerebrovascular disorder is often found to be higher in the unrecovered cases of psychoses of later life (cf. Strecker *et al.*, 1931) this study has shown that the mere presence of cerebral vascular disease, especially without demonstrable dementia, is of no prognostic significance so far as endogenous depression is concerned.

It would seem that this earlier occurrence of cerebrovascular disorders with psychiatric disturbances encountered in Africans may be precipitated by associated factors, e.g. malnutrition, avitaminosis, and recurrent malaria infection. On the other hand, Meyer and McLardy (1950) quote the findings of Hirsch (1945), who was unable to demonstrate any correlation between arteriosclerosis and age in 2,051 brains, and no demonstrable sign at all of arteriosclerosis in 9.5 per cent. of autopsies in 400 old men over the age of sixty-five. Therefore, in most of these cases, the matter of physical or psychic primacy is of interest but obscure.

Hirsch was reported to have concluded that arteriosclerosis is a malady, not of old age, but of civilization. Evidence of any such assumption seems to me, after examining the problem in the light of cultural differences, extremely unsatisfactory and questionable. It is my impression that cerebral arteriosclerosis and essential hypertension occur among the primitive and relatively isolated rural population as often as they do in civilized and cultured African communities, although it may not have any correlation with age. Hirsch's generalization would have been much more appropriate in respect of coronary arterial disease.

However, one fact which stands out clearly in this study is that when the two cultural groups are compared—the rural illiterate African patients and the urbanized (and especially professional) patients—the psychological aspects of these disorders recede completely into the background in the former. Emotional and psychological factors play a much more obscure role in the genesis and dynamics of the symptomatology of the rural Africans, in contrast to the urbanized arteriosclerotic and hypertensive patients. Over 95 per cent. of the latter knew that they had had hypertension and had lived for years under the burden of anxiety with regard to the ever-increasing fear of "stroke" and cardiac disease before further emotional and psychological factors played an important role in causing the depressive illness often with a neurological condition. Also most of these latter group of patients were involved in protracted domestic difficulties concerned with family, financial, professional problems or other aspects of social life.

Psychological tensions and neurotic manifestations (e.g. hyper-irritability, insomnia, explosive temper, neurasthenic symptoms) are often widespread in the literate urbanized hypertensives. These symptoms which are much more of premonitory symptoms of endogenous illness nearly always antedate the

major and more recognizable signs and symptoms of cerebrovascular disorders.

Psychotherapeutic measures have therefore been found to be an important adjuvant to the total management of most of these patients. Environmental manipulation has proved useful both as diagnostic and therapeutic measures.

METHOD

Each patient was first examined in the "Day Hospital" Clinic where his full social and clinical history was taken, appraisal of neurological and mental findings were recorded with a description of the fundi. All patients who showed deep and sustained depression, clear-cut history of cyclothymic fluctuations of mood, and focal neurological signs and symptoms were selected for study in the absence of demonstrable dementia.

Every patient was interviewed daily and the neurological signs and symptoms were assessed and recorded thrice a week in relation to the nature, character, and course of affective disturbances. Appreciable degree of lability of blood pressure found in most of these hypertensive patients made for an easy assessment of the role of psychological and environmental factors in the relationship between the immediate rise of blood pressure and the psychiatric symptoms. In all the patients, head injury, intracranial tumour, G.P.I., myxoedema, pernicious anaemia, and toxic-infective processes were ruled out as the primary aetiological factors, since the incidence of some of these conditions is higher in the old age group and affective reactions may be symptomatic of these conditions.

Wherever possible, exhaustive examination of the near relatives of the patients was carried out. Forty-five relatives (mostly siblings) were examined in this way. Basal blood-pressure readings were made and several intravenous barbiturate tests were also carried out after casual blood-pressure readings had been made. In addition to these tests, intravenous histamine acid phosphate—nicotinic acid injections were given and all reactions—physical, including blood pressure, and mental—were evaluated. A "normal" control was set up and, under similar conditions, these tests were repeated.

There is definite evidence that the relatives of hypertensive patients showed excessive range or lability of vasomotor reaction. Greater number of side-reactions, e.g. tension, headache, various subjective feelings, and hypotension, were much more common ($66\frac{2}{3}$ per cent.) in the near relatives as against the 20 per cent. in the control group. In eleven of the relatives the fall of blood pressure approached dangerous levels. In the intravenous sodium amytal tests the drop in the diastolic pressure was more considerable in the relatives than in the control group.

Occupational therapy was made use of extensively in observing some of these patients. It has proved of value in an observation of this sort, not only from the therapeutic standpoint, but as an early indicator of variations in behaviour, including the attitude of the patient, fluctuations in interest, attention and adaptability, and, to a slight extent, prognosis. The criteria of full recovery in this study are based upon the total disappearance of mental symptoms (not of premorbid traits) as well as remission of physical signs and symptoms over a period of 12–18 months.

From the treatment angle, all our patients who presented an affective syndrome with a history of either cyclothymic fluctuations for a number of years or of depressive constitution received E.C.T. and intensive psychotherapy. In addition, the hypertensives received hexamethonium while the arteriosclerotic

group responded well to intravenous glucose-aminophylline-nicotinic acid therapy given twice a day for four to six months.

ILLUSTRATIVE CASES

Brief case summaries are given below to save space. Two from each group are selected because of their illustrative features.

Hypertensive Group

There is very little to add to Mayer-Gross's (1937) classical description of the syndromes and symptoms associated with hypertension. The neurological and psychiatric syndromes resulting from hypertension in our study were characterized by their episodes, fluctuation, and certain degree of reversibility. Psychotic episodes were characterized by profound clouding of consciousness which may last several days during which hallucinations and delusions may appear. Prodromal symptoms—hysterical outbursts, subjective sensations and excitement—were frequently observed.

While it is feasible that the well-recognized symptoms are explicable on the basis of vascular dysfunction (cf. Guttman), a great deal of the psychological disorder associated with cerebral hypertension may conceivably represent reaction to an intrinsically neuro-organic situation to which the personality changes may be the fundamental ones.

The following cases, taken from the ten selected patients observed at Aro, may illustrate the role of inherited predisposition and psycho-reactive factors in determining the clinical picture and course of illness in spite of the associated organic features.

Case 1. (No. 9), Male, aet. 47. Yoruba Civil Servant. Married with five children. Family history showed that his father, although he never broke down, had cyclothymic fluctuations of mood for years; an uncle committed suicide during a depressive illness, and a sister has had two attacks of hypomania. Father was of pyknic features although the patient is of asthenic build. In the family as a whole there is a predisposition to mood swings.

No previous attack of depression but patient had always been rather talkative and excitable and his wife described him as being of cycloid personality. While in England in 1950, he spent money rather lavishly, had a sudden inspiration to buy antiques on which he spent several hundred pounds, and ended up in making extravagant financial deals without mature deliberation. Two years earlier, he had a short spell of insomnia in which he woke up between 2-3 a.m. every day for about six weeks and insisted on discussing excitedly current problems with his wife in bed.

In 1950, while in England, he was examined while taking out a heavy insurance policy and was told for the first time that he had high blood pressure. Since then he became obsessed with the idea of sudden death from "stroke" and finally developed a host of neurasthenic symptoms for which he has been taking several pills a day and dieting vigorously.

His present illness began with depression a month before he was seen. Following financial and domestic difficulties involving his honour as a person and his professional dignity he became suicidal but was too depressed to make an attempt.

Findings: He showed complete psychomotor retardation, slightly suspicious. Episodes of vestibular disturbance, headache, insomnia, severe paroxysmal anxiety associated with B.P. lability of 260/130 to 200/120. On two occasions he had confusion—delirium syndrome and after the hypertension had been controlled he slipped into a depressive stupor. The latter condition, which finally cleared up with E.C.T. was followed by feelings of well-being, exuberance of spirit, possibly a slight hypomanic phase.

During the psychotherapeutic sessions that followed, blood pressure fluctuations and attacks of vestibular disturbance were noticed during the discussion of the psycho-reactive factors which were so prominent in the genesis of the present illness.

Case 2. (No. 7), Female, married, aet. 45. Essential hypertension with sustained depression and retardation. Complained of irritability, inability to concentrate, lack of energy and severe depression, fainting spells, dizziness and insomnia. Menopausal symptoms were active on admission. Signs and symptoms subject to emotional fluctuations. At the height of emotional difficulty with a psychopathic husband patient had two encephalopathic attacks. Diastolic pressure was never below 130 mm. Hg.

Family history revealed that one brother had paranoid and depressive illness for two years during which he had "epileptiform fits". The brother who was examined later had B.P. of 210/110. Father died of "apeta" (? coronary thrombosis) and mother, who was arteriosclerotic, aet. 66, had a short manic illness just after the birth of the brother. She (mother) was of pyknic habitus and syntonik temperament. Patient's sister aet. 48 had slight arteriosclerosis and hypertension of 200/120 without overt symptoms. She was described as being temperamental and has had "mood spells" for years.

At puberty patient had a mild depressive illness and lack of appetite. Otherwise she had been in good health all her life but started to have menopausal symptoms a year before she was seen. Later, her husband who was a lawyer began to associate with young women and was keeping late hours. This upset the patient (who was of strong religious and strict outlook) a great deal. She suddenly found one day that the whole of her left side became weak after a severe bout of headache for a day.

Patient made good improvement. At the outset she became emotionally unstable, manifested multiple hysterical and fleeting hypochondriacal symptoms in relation to her family difficulties. Preliminary treatment of her high blood pressure and hormone treatment of her menopause followed by E.C.T. improved her mental and physical state. Psychotherapeutic situation which included the husband played an important part of the treatment programme. The husband finally showed some consideration and latterly has co-operated. Patient was discharged home fully recovered and has been free of mental symptoms for over a year. B.P. remains between 160/110 and 140/100. She was of rigid, obsessional personality and attended church "very regularly".

Whilst in the majority of cases, syndromes of confusion and marked but fluctuating affective disturbances as well as profound degree of reversible personality changes may dominate the picture, nevertheless three cases have been seen in which the clinical picture is principally constituted by psychotic disorder and progressive disintegration of the personality, usually seen in functional psychoses and differing from cerebrovascular degeneration of senile and arteriosclerotic disease. One of these was mixed psychosis with depressive and hypochondriacal features; the others showed admixtures of schizophrenic and depressive reactions.

Arteriosclerotic Group

A difference of opinion exists as to the nature of the concept of cerebral arteriosclerotic psychosis, the criteria for diagnosis and its psychopathological features. It would appear rather difficult to differentiate some of the features of the cases encountered here possessing definite pattern of pre-morbid personalities (predominantly of the syntonik type) from true, late appearing or masked, endogenous depression.

The syndrome which goes to make up the condition is constant and characteristic in over 60 per cent. of our cases. Affective disorder, especially depression, was constant and deep as opposed to the shallow and fleeting depressive episodes associated with various organic diseases of the central nervous system. In almost 40 per cent. of this group of late endogenous forms of depression earlier indications of such mental reaction which only became patently manifest at involutionary age or precipitated by cerebral organic disease were found.

On the basis of the usual criteria (focal neurological signs and symptoms, marked fluctuation of neurological and psychiatric symptomatology in an organic setting, relative preservation of the personality, especially a history of a previous vascular hazard), for the diagnosis of cerebral arteriosclerosis, the present observation would seem to relegate these "standard" criteria to one of arbitrary nature. Despite this, these rigid criteria were maintained in the selection of our patients, otherwise questions as to their diagnostic and prognostic import would assume a subsidiary position.

Eight of our series of twelve patients manifested profound depression; two showed manic symptoms while two were mixed, with schizo-affective

features. Therefore in a large proportion of the present clinical material, the earliest symptoms are manifestly depressive (profound and sustained) and in their quality and character they show essential departure from the usual. On the other hand, the residual neurological signs and symptoms were of mild and fleeting nature and *there were no clinical signs of dementia*.

Here, then, is a factor in the manifestation of arteriosclerotic psychosis which would seem to merit more general recognition, especially as Roth (1955), with his unique experience, observes this "overlap between the disorders" and considers it to be an important factor "since it is this problem that has the greatest bearing upon the prognosis and treatment of mental disorder in old age". Furthermore, its recognition would seem to be of extreme importance, because it prevents an undue emphasis upon a certain *somatic vulnerability* (Meyer, 1936) as regards the pathogenesis, which, however, under the stress operative upon the psychic level, may conceivably become clinically manifest as described.

Case 3. (No. 8), aet. 49. Cerebral arteriosclerosis. The findings consist, *inter alia*, of right hemiparesis, confusional state for 3 days, receptive dysphasia, moderate hypertension and peripheral arteriosclerosis. B.P. 170/120.

Family history—father who is dead had asthma since childhood. One brother alive and well, aet. 52, suffered for the past ten years from periodic skin disorder "which comes and goes" and which two of the latter's children have "inherited". A married sister (through whom the patient reached us) is alive with neurasthenic symptoms and severe degree of obesity. She is cyclothymic and once had a skin lesion simulating disseminated lupus erythematosus which responded to hypnosis. She is also moderately hypertensive. The youngest sibling, also a married woman aet. 40, had puerperal psychosis of a depressive nature at 30 and she has only partially recovered. Paternal uncle had one attack of manic-depressive psychosis at 48 but has been free from an attack for sixteen years.

Patient's premorbid personality was cyclothymic but he was on the whole a sociable, generous and happy man. Had vertigo for two years and this worried him considerably. Since the death of his son he has suffered from insomnia and on losing his job became morose and had his first attack of "stroke", resulting in right hemiparesis and dysphasic symptoms. Later, the depression deepened and he became very retarded for several months.

Many unmistakable features of cerebrovascular disease—vertigo, hemiparesis, dysphasia—cleared up completely within five months with treatment and the depression cleared up later with E.C.T. The patient made a complete recovery and has since got a new job and was reported to be working satisfactorily.

Examining this case we see a family with interesting psychosomatic problems (atypical skin lesions, asthma), manic-depressive psychosis, and cerebral vascular disorder. Emotional problems were particularly common in this family and the various ways of their expression are of great interest. The common denominator may be a constitutional factor.

Case 4. (No. 5), Male civil servant, aet. 47. Cerebral arteriosclerosis. Patient complained of headache, insomnia, episodes of confusion for two years.

Father had a depressive illness about the same age as the patient and the development was similar in both cases. Father was irritable and agitated for months before he started to have what would seem to be attacks of depressive stupor according to the patient's description. Five younger siblings are well. Paternal uncle has a depressive constitution and is known in the family for his gloomy outlook.

Patient had no neurotic traits in childhood but suffered from chronic abdominal pain and recurrent upper respiratory catarrh when he was a boy. These conditions disappeared around puberty.

His present illness had started two years before he was seen when he noticed that he was having frequent attacks of dizziness; he also noticed constant irritability, lack of concentration and early morning waking as prominent symptoms. On one occasion he described an attack of severe confusion and disorientation which finally ended in "paralysed right leg". His condition improved gradually without treatment but the recent relapse, of four months duration, has been very troublesome and has been associated with severe agitated depression. Demonstrable psychological factors were of importance at the outset. "Alleged official irregularities and bribery" provoked considerable anxiety and initiated the ensuing insomnia. Later, feelings of "tightness in the head" and various somatic symptoms became prominent.

When first seen he was very agitated and depressed. No demonstrable physical signs, apart from peripheral and retinal arteriosclerosis and B.P. of 150/115. He slept poorly and hardly ate anything. The more general symptoms, e.g. stupor and irritability, which could under the circumstances be assigned to the diffuse vascular condition were latterly appraised

as manifestations of endogenous depression. Later, in the course of his illness, he had intracerebral haemorrhage with a clinical picture of mild pseudo-bulbar palsy from which he recovered sufficiently to have electroplexy.

Patient recovered with combined psychotherapy, electroplexy, and intensive vitamin-aminophylline intravenous injections and has remained well for nearly eighteen months.

DISCUSSION

There is definite indication that certain types of mental disorder associated with diseases of the cerebral arteries in old age are often causally related to endogenous factors; some cases would seem to be reactivation of psychiatric disorders which had taken place earlier in life or provocation of latent disorder which had hitherto failed to become manifest. In three of our cases involutional depression was antedated by pubertal depression of moderate severity which cleared up spontaneously after varying lengths of time.

Even though in some cases profound apathy rather than true depression was present, endogenous depression has been proved to be the most likely diagnosis. Although the history of a previous attack of affective disorder is not in itself sufficient to clinch the diagnosis, the character and qualities of the psychotic reaction are sufficient to remove them from the category of those which are merely symptomatic of organic diseases. However, Smith (1954) has warned that several organic syndromes may closely simulate involutional melancholia.

Other neuro-organic conditions were also excluded. For example, Stengel (1943), quoted by Smith (1954), also stressed the clinical similarity between some cases of pre-senile dementia and involutional agitated type of disorder at some stages of their manifestation. Smith again referred to Noyes (1947) and Ewen (1947) who both showed the difficulties that one may encounter in the differential diagnosis between involutional melancholia and cerebral arteriosclerosis.

However, Smith (1954) seriously considered the possibility "that in some cases the organic conditions merely acted as precipitating causes on a soil already prepared". He finally concluded, "Put in another way, the affective manifestation of anxiety and depression might seem to be some sort of pre-formed cerebral function occurring in the personality type and potentially manifesting itself when the right precipitating factor came along".

If we could accept this view with certain reservation, we should be able to differentiate such cases and exclude them from the heterogeneous mass of clinical material generally labelled "old age organic psychoses", apparently of unfavourable prognostic significance.

With the onset of headache in our hypertensive patients, as a cerebral attack approaches, the mental picture as a rule changes rapidly. A depressive or maniacal state may ensue at once, but the most common disturbance is some phase of mental confusion and a mild delirium. Marked anxiety and fear are not uncommon during an attack. In some cases the headache had at times been entirely replaced by depression, stupor, automatism, compulsive acts and irritability. The latter is a symptom which has featured prominently in all the cases belonging to both groups of cerebrovascular disorder.

The psychiatric manifestation in arterial hypertension can therefore be divided into four groups: those occurring (*i*) during the prodromal period; (*ii*) with the encephalopathic attack; (*iii*) as equivalents; and (*iv*) as associated phenomena. This last group is of importance clinically as it includes late appearing psychiatric conditions like involutional mental changes, or manic-depressive psychosis.

For this necessarily brief account of psychiatric syndromes associated with essential hypertension, the point of importance is that it would seem that hypertension, after initiating the vicious circle, fails to account in itself for its influence on the total clinical picture. Six of our ten patients recovered fully (i.e. to the status quo ante), one died of intracerebral haemorrhage, two are recurrent and one was unchanged.

There is thus a striking argument in favour of constitutional and psychological factors in the determination of the entire psychiatric picture, especially those features which one would normally consider purely endogenous.

In considering cerebrovascular psychiatric syndromes as a whole, wherever constitutional factors appear to be contributory or significant, the physio-pathological condition seems to render the patient particularly vulnerable to psychological stress. Since it has been shown by Palmer *et al.* (1943) that it is unlikely that more than a fifth of persons with advanced arteriosclerosis have psychotic breakdown, hence the importance of genetic-constitutional factors and other pathogenetic agencies in those who manifest psychosis.

In all but two of our arteriosclerotic cases the psychiatric symptoms were present from the time of the onset of the disease or within a short period after the appearance of physical signs and symptoms. In those two instances there was a considerable interval of time before physical signs and symptoms appeared. In both of these patients hemiparesis developed gradually.

In another case in spite of the fact that sustained, deep, and agitated depression antedated the onset of severe physical signs and symptoms, the prognosis was bad. There was progressive deterioration and death. This supports Smith's view that from a practical consideration, full clinical assessment of associated physical anomalies in most of these disorders is obviously of the greatest importance.

The present observation strongly suggests that possibly emotional states produce, among other things, cerebral circulatory alteration which, in the presence of disease, may further cripple functions already impaired, also it is conceivable that in many cases of cerebral arteriosclerosis, regardless of the priority of the mental disturbance or of the physical manifestations, there is certainly a circle of abnormality established constitutionally, the arcs of which are composed of both groups of factors. Such views, though not conclusively proved, at any rate make it more easy to understand the disparity between the degree of mental picture and severity of physical manifestations, and the role of inheritance not only in determining *possible genetic relationship between cerebrovascular degeneration in later life and endogenous psychosis (especially of manic-depressive variety)* but also in assessing the prognosis of these disorders. The concomitance between other possible psycho-physiological connections as yet unknown may well play some significant role.

With regard to these possible constitutional and biological connections, all writers have agreed on the (alleged) rarity of endogenous depression and the predominance of mania in the coloured race. It would seem that environmental and cultural factors play an important role in the high incidence of mania and relative absence of depression below the age of 40 in the coloured race. However, clinical data obtained in Nigeria over a period of five years tend to point to (i) relative rarity of "pure" forms of constitutional depression below the age of 40; (ii) relative increase over the age of 40 and (iii) high correlation between this psychosis and cerebrovascular disease over the age of 45. This immediately raises the question of constitutional or racial liability for manic-depressive patients not only to develop cerebral vascular disorder but also to manifest the

depressive component at the time of the association of both groups of diseases.

With the aid of those which constitute the present material, therefore, Rothschild's emphasis on the psychosomatic connections between genetically-determined personality patterns and such diseases as arteriosclerotic psychosis is justified, and at the same time, also, Roth's observation that arteriosclerotic and senile changes in the brain are "not necessarily relevant to the psychoses developing in old age" is confirmed from a prognostic standpoint.

Out of the series of twelve arteriosclerotic patients selected because of the endogenous features of their psychoses (and fairly definite premorbid psychobiological reactions) in an organic setting, five patients who showed profound and sustained depression with apathy and the two manics recovered fully with E.C.T. Out of the remaining five, three who showed neurasthenic symptoms and involutional type of depression improved sufficiently to go home; one with mixed psychosis and another with schizoid-psychopathic features became progressively worse. Evidently, in the recovered cases, the presence of cerebrovascular disorder has been prognostically unimportant, except as to the risk of fatal cerebrovascular hazards.

In most of them the presenting organic signs and symptoms (e.g. confusion, giddiness, paraesthesia, transient hemianopia, and other focal cerebral attacks) were mild, non-progressive and fleeting. In most of our cases these prognostically unfavourable signs and symptoms disappeared altogether within three to six months after intensive physical (drug) treatment, leaving an uncomplicated picture of endogenous or involutional depression. It is obvious that in these cases, as in the cases of the other authors referred to, the clinical picture with its special evolution cannot be understood wholly in the light of a special morbid process but in the light of constitutional factors as well as psychological circumstances, and it is especially in regard to the latter aspects of the problem that these observations furnish a document of interest.

A feature not usually mentioned but present in this work is the presence, in their order of incidence, of coronary arterial disease, asthma, various skin lesions, peptic ulceration, and periodic headaches in the near relatives of the patients and the relatively low incidence of these disorders not only in the control group but also in the patients themselves. If this observation is worth anything, one is tempted to say that most of these patients come from a stock which has the capacity to utilize *somatic compromise* to offset damage to higher psychic or organic functions, but they (patients) themselves lack the inherent capacity to ward off cerebral vascular damage as evidenced by their failure to develop compensatory psychosomatic illness of a well-defined character.

It is hoped that the psychosomatic aspects of these disorders will be studied more in detail within the next few years to facilitate statistical analysis.

The main point raised in this paper is an attempt to show that the physiopathological theory of cerebrovascular disorder is associated with the hypothesis of the influence of the factor of inheritance. I feel that the two lend each other "moral" as well as logical support, and emphasize the wider scope of the psychosomatic aspect of the problem, thereby strengthening my view that only when we have exhaustively scrutinized the personal and family record with a negative result can we allow cerebral vascular dysfunction *per se* to be the *causa causans* of the psychiatric syndromes.

It seems as if racial (biological) and/or environmental factors in this group of people under observation enhance this "overlap" between, or merging of, these psychoses. That means that in other "racial" groups the "overlap" may

be less significant in that organic syndromes may relegate the involutional phenomena or endogenous depressive reactions to the background.

The results of this inquiry, obtained as they are from such meagre data, have been evaluated with great caution. Despite the obvious defects of the investigation, the results which are based on unprejudiced assessment of the clinical facts emphasize the need for further research into the genetic-constitutional relationships of these disorders.

SUMMARY

Certain aspects of psychiatric syndromes associated with hypertensive and arteriosclerotic disorders in African patients are described. Each case was taken as a separate problem and examined without theoretical prejudice. Environmental and psychological factors—such as current emotional problems, psychic trauma—received the same consideration as internal factors based upon the properties of inherited predisposition. Clinical reasons for suspecting constitutional and personality factors in aetiology and for considering the syndromes observed as endogenous were discussed.

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DIFFERENTIAL EFFECTS OF LYSERGIC ACID AND SODIUM AMYTAL ON IMMEDIATE MEMORY AND EXPRESSIVE MOVEMENT

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INTRODUCTION

THIS investigation deals with relationships of sodium amytal and lysergic acid (LSD) to measures of immediate memory and expressive movement as a function of exposure time. Significant differences in learning and expressive movement had been obtained previously between neurotics and schizophrenics (3, 4). The extent of these differences posed the problem why memory disturbance, as in the test, is clinically not more obvious in schizophrenics than in neurotics. It was thought that accidentally favourable conditions had been used, either in difficulty of task or in exposure time, to produce the results. The decision was taken to vary the exposure time in a pilot experiment, using LSD in normals to represent schizophrenic-like symptoms.

Since the test used permits measurement of expressive movement concurrently with memory the *first aim* of the present study was extended accordingly. It served to investigate the effects of various exposure times on immediate memory and expressive movement under the conditions of LSD and amytal. The *second aim* was to test the hypothesis that test scores of normals given LSD, as against amytal, vary in the direction of the previously established norms of schizophrenics. The lack of a placebo condition in the present experiment does not permit the testing of differences from a normal standard. The *third aim* was simply to validate the test used, a recall version of the so-called Figure Reconstruction Test (FRT), against the previously used learning version of the same technique. For this reason, more scores will be analysed than actually required.

The experimental use of LSD to obtain schizophrenic-like symptoms appears adequately justified by the literature. If LSD effects are considered to be an expression of "neuronic hyperexcitability" (7), sodium amytal, as a sedative, may be expected to produce opposite effects on many test scores. Amytal, among other drugs, reduces the clinical manifestations of LSD intoxication. This drug was, therefore, expected to maximize the differences. Assessing the literature, it may be said that LSD does not necessarily affect memory significantly, as measured by conventional tests (Savage, 11; Sloane and Lovett Doust, 13). From these studies, when compared with the experiments of Jarvik *et al.* (9), it may be deduced that up to 50 micrograms of orally administered LSD does not impair immediate memory significantly. After 100 micrograms, however, a certain amount of memory impairment was

demonstrated (9). This finding, being inconsistent between various tests, requires confirmation. As far as amytal is concerned results are equally conflicting. Some hours after an hypnotic dose was given, memory for digits was found to be significantly impaired (Goodnow *et al.*, 8), whereas serial learning of a 15-syllable list was not impaired and recall only under specific conditions (Felsing *et al.*, 6). Both LSD and amytal may, therefore, under specific conditions only, be expected to impair memory. But there is nothing in the literature to suggest that the two drugs might have differential effects, with the exception of digit span which was found to be impaired after amytal and not after LSD. In the present experiment, the position is taken that error should be higher for LSD on the grounds that their clinical representatives, the schizophrenics, of all functional categories investigated scored the highest error (3).

As far as unintentional expressive movement is concerned no results bearing directly on the present situation are known. Judging from other types of tests like the Bender-Gestalt Test (1), or steadiness (1) and finger dexterity tests (10), increased variability may be expected after LSD as against greater steadiness after amytal.

Equating LSD effects with those of schizophrenia, the following *predictions* in terms of test scores used may be made on the basis of previous experiments quoted above (3, 4).

Score	Previous Result	Prediction for LSD as against Amytal
Rotation Error	Schizophrenics higher score than neurotics and normals	Higher score for LSD
Distance Error		
Size Variability (within and between shapes)	Schizophrenics higher than neurotics and normals	Higher score for LSD
Distance Variability (pattern and range)	Tendency for schizophrenics to score higher only after a certain amount of practice*	No significant difference, or higher score for LSD
Spread (corrected for mean distance)	Consistent though not significant tendency for schizophrenics to score lower than neurotics, particularly hysterics, and normals	No significant difference, or lower score for LSD

* This is one of the reasons why three FRT sets were given before the experiment started.

When making these predictions, it must be kept in mind that in the present experiment stimuli changed with each exposure (immediate recall). In the previous studies, from which the predictions are derived, the stimulus was kept constant throughout a number of trials (learning).

METHOD

1. *The Figure Reconstruction Test (immediate recall version)*

This test, referred to as FRT, has been demonstrated reliably to discriminate between normals, neurotics and psychotics, when used as a learning test. In the present experiment a recall version of the FRT is used, where no stimulus is shown twice. One test set consists of ten stimulus patterns, an example of

which is demonstrated in Figure 1. The polargraphic network shown is for demonstration only and not actually present in the stimulus exposed to the S, except for the central circle. Each of the ten stimulus patterns of a set contains the five geometric elements: semi-circle, circle, triangle, square and oblong.

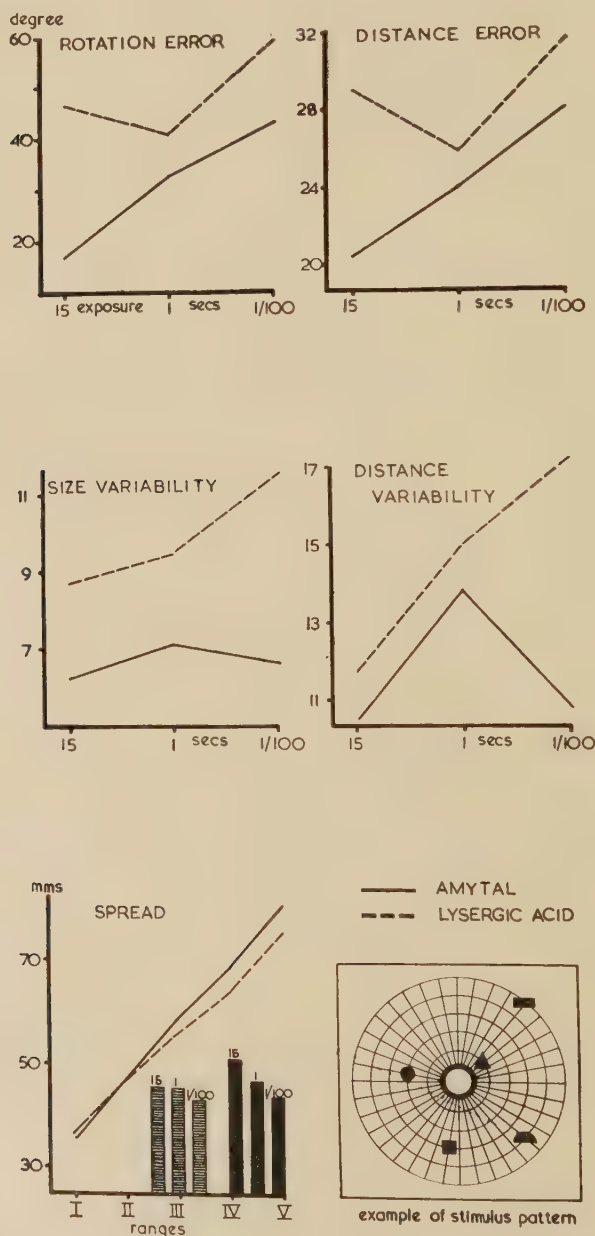


FIG. 1.—The relationship of error and expressive movement scores to LSD and amytal, as a function of exposure time. An example of test patterns, from which scores are derived, is shown. Scores are ratio scores, except for rotation error.

Over the ten patterns of one set, these shapes are distributed in such a way as to achieve a balanced axial and radial distribution relative to the intersection points of the network.

The central circle is shown on both the stimulus cards and the reproduction sheets, where it serves as a reference point for scoring. The *instruction* is to reproduce *all* five test shapes around the central circle, as seen on the screen. Stimuli are drawn in indian ink on a square white card board of 23 cm. side length. The reproduction sheets are of the same size. *Exposure* takes place on a black screen, at a distance of 65 cm. from the S's eye. Inspection occurs through an ordinary compur shutter, which provides the desired *exposure times* of 1/100, 1 and 15 secs. duration. During the exposure the stimulus only can be seen on a uniform black background. Before the experiment each S was given a full *trial session*, comprising all three exposure times, to reduce practice effects and to familiarize him with the task.

2. Test Scores

Scores have been described previously in full (3, 4). They are as follows, (a) and (b) being measures of memory, (c) through (g) measures of expressive movement.

(a) *Rotation error*: mean axial displacement of test shapes in degrees.

(b) *Distance error*: mean radial displacement of reproduced shapes from the stimulus position in mm., divided by the mean distance of shapes from the centre to correct for variations of drawings in distance. This score varies slightly from the one originally used.

(c) *Size variability (between shapes)*: mean size deviation of the five reproduced shapes from their mean, divided by size (correction for size). "Size" is the base length of reproduced shapes in mm. Variability scores are computed separately for the ten trials pertaining to one set and then averaged.

(d) *Size variability (within shapes)*: identical with score (c), except that deviations are measured within the five categories of shapes, for example within all ten triangles reproduced in one set. Corrected for size.

(e) *Distance variability (between pattern means)*: mean distance deviation of the ten patterns from their mean, divided by the respective mean distance (distance correction). "Distance" of a shape is measured in mm. away from the centre of the reproduction sheet.

(f) *Distance variability (within ranges)*: similar to score (e), except that computed separately for the five "ranges", or distances from the centre at which S's choose to reproduce. Corrected for distance.

(g) *Spread (between test shapes)*: mean deviation in mm. of the five test shapes from their mean distance score. Computed separately and averaged over the ten trials. Distance correction applied.

In Figure 1, the two size and distance variabilities have been combined, as the respective results were very similar. The scoring system presented is comprehensive, as the test is still under construction. It will be noted that all expressive movement measures are corrected for the scores from which they were derived. The basic scores "distance" and "size" have originally been used as separate scores. They have, however, so far (the present study included) appeared to be relatively poor discriminators.

3. Subjects

Six subjects, all medically qualified, were used. The age varied between 26 and 38 years. There were five men and one woman.

4. Drug Administration

LSD-25 was given intravenously, 100 micrograms dissolved in 5 c.c. water and injected over a period of one minute. Testing started one hour after the injection. Sodium amytal was injected using a dosage of 0.5 mg. per kilogram body weight per c.c. at a rate of one c.c. every 40 seconds until "sedation threshold" (Shagass *et al.*, 12) was obtained. Further injection continued till S was slightly drowsy, or fast barbiturate-EEG activity appeared to have reached maximum amplitude. The total dosage, with the injection times stated in brackets, varied with the S's as follows: 0.40 gm. (30 minutes), 0.40 gm. (20 minutes), 0.38 gm. (20 minutes), 0.45 gm. (25 minutes), 0.50 gm. (20 minutes) and 0.40 gm. (35 minutes). Testing began 30 minutes after the injection.

5. Design of the Experiment

With the introductory trials, two drugs and three exposure conditions, six test sets of ten stimuli each were required. The construction of parallel forms of the FRT is easy and reliable. Drugs, tests and exposure times were then systematically varied so as to minimize errors due to variation in test sets and practice. Between the two administration dates a period of one week was allowed to pass.

RESULTS

An analysis of variance was performed separately for all test scores, as exemplified in Table I.

TABLE I
Analysis of Variance for Rotation Error Score

Rotation Error			d.f.	s.sq.	m.sq.	F-ratio	Significance (Per cent.)
Exposures	..	..	2	2,312.055	1,156.028	7.305	1
Drugs	..	..	1	2,952.111	2,952.111	18.684	0.1
Interaction D × E	..	..	2	722.391	361.196	2.282	NS
Between classes	..	..	5	5,986.557			
Within classes	..	..	30	4,747.665	158.256		
Total	..	..	35	10,734.222			

The mean variances for exposures, drugs and their interaction were tested against the within classes variance. As a result of the design, the interaction terms were expected to be insignificant. Subsequently, independent t-tests were carried out, where applicable or advisable. The results are summarized in Figure 1 and Table II, which states the F-ratios and significance. Table III contains the means of all test scores used, separately for drugs and exposure times.

TABLE II

Results of Analysis of Variance similar to that shown in Table I

F-ratios	Exposures	Drugs	E × D
Rotation error	7.305 (1%)	18.684 (0.1%)	2.282 (NS)
Distance error	3.503 (5%)	6.423 (5%)	1.239 (NS)
Size variability (between shapes)	0.825 (NS)	10.526 (1%)	1.168 (NS)
Size variability (within shapes)	0.310 (NS)	5.565 (5%)	0.108 (NS)
Distance variability .. (between patterns)	3.185 (10%)	8.074 (1%)	0.621 (NS)
Distance variability .. (within ranges)	2.016 (NS)	4.910 (5%)	2.228 (NS)
Spread	3.590 (5%)	3.998 (10%)	0.830 (NS)
(between test shapes)			

Degrees of freedom: exposures 2/30, drugs 1/30, E × D 2/30.

Significance levels in brackets.

TABLE III

Means of all Test Scores used, separated for Drugs and Exposure Times

	Lysergic Acid			Sodium Amytal		
Exposure time in seconds	15	1	1/100	15	1	1/100
Rotation error	44.7	40.7	59.2	17.0	32.8	42.3
Distance error	29.0	26.0	31.5	20.5	24.0	28.3
Size variability (between shapes)	9.3	10.5	13.0	7.2	7.8	7.0
Size variability (within shapes)	8.2	9.3	10.2	5.7	6.3	6.2
Distance variability .. (between patterns)	12.7	15.7	16.7	10.3	13.5	11.8
Distance variability .. (within ranges)	10.5	11.3	12.3	10.0	11.2	10.0
Spread	21.8	21.0	19.0	27.7	22.8	20.5

1. Analysis Between Drugs

Error scores were found to be significantly higher for LSD as compared with amytal. The superior result of rotation over distance error is in accordance with the relative sensitivity of these scores in discriminating between normals and abnormals (3). The two drugs were further significantly and consistently different as regards all four *variability measures*. *Spread* did not significantly differentiate between the drugs. It will be noticed, however, that the F-ratio falls near the 5 per cent. level of confidence. Moreover, the contraction effect predicted for LSD, is in the correct direction and very similar to the corresponding test behaviour of schizophrenics, whose spread score tended to be low when compared with other clinical groups.

2. Analysis Between Exposures

For the error scores, differences between exposures were significant, as expected, but not nearly to the same degree as between drugs. Furthermore, LSD produced a new phenomenon in memory in that the mean error was higher after the 15 secs. than after the one-second exposure. In the case of rotation error there was no overlap between the two drug conditions and five of the six S's showed the paradox described. Comparatively speaking, this effect of the drugs on presumably highly intelligent normals was such that the

15 secs.-LSD performance was significantly *worse*, and the 15 secs.-amytal performance significantly *better* than corresponding performances of either neurotics and schizophrenics on the same task.

Independent t-tests for related measures yielded the following results (five d.f. throughout).

Exposure seconds	..	..	..	15	1	1/100
Rotation Error	..	..	..	3.821 (2%)	NS	3.085 (5%)
Distance Error	..	..	..	2.442 (10%)	NS	NS

It appears from this that the greatest differences were, if anything, obtained for the long exposure time. However, taking rotation error alone, as the most reliable discriminator, a non-linear relationship may be seen.

As far as the *variabilities* are concerned no significant differences were obtained between the exposure times, as seen from the F-ratios (Table II). In view of the larger differences in the means of all four measures at the 1/100 sec. exposure it was decided to compute independent t-ratios for related measures with the following results (five d.f. throughout).

Exposure seconds	..	..	..	15	1	1/100
Size variability (between)	..	..	..	NS	NS	11.617 (0.1%)
Size variability (within)	..	..	..	NS	NS	6.324 (0.1%)
Distance variability (pattern)	..	..	..	NS	NS	3.061 (5%)
Distance variability (ranges)	..	..	..	NS	NS	6.998 (0.1%)

Highly significant differences were obtained for the 1/100 sec. exposure only.

For *spread*, differences between the exposures were significant at the 5 per cent. level of confidence. In this case, results for the one and 1/100 sec. exposures were insignificant. For the 15 secs. exposure, however, a t-ratio of 4.032 (five d.f.), significant at the 1 per cent. level, was obtained.

DISCUSSION

All predictions concerning the differential effects between LSD and amytal have been confirmed. This may be said to support the view that LSD acts, under the limitations of the present experiment, in a manner consistent with and, if anything, stronger than that of schizophrenia. It may be added, that the outcome of the experiment would not be consistent with the test behaviour of various neurotic categories.

The most important difference in results between LSD and amytal is that the drugs have *specific* effects on memory, and do not just produce a general impairment of function, as might be found for example in a toxic state from many causes. The dependency of LSD memory impairment on relatively long exposure time may in part explain why, in the original experiments (3), schizophrenics learned more slowly than neurotics and normals. At that time, an exposure of five secs. duration was used, which may be sufficient for detrimental effects to occur, as the following introspection of the senior author suggests.

After the application of LSD and during the 15 secs. exposure the black test shapes on the white stimulus cards were soon surrounded by a yellow

halo. With exactly four seconds of the exposure gone, test shapes became wobbly, jumped and completely left their position to march around the stimulus card in an orderly procession. This phenomenon could be repeated with every exposure, but never during the short exposure times. Similar introspective reports were spontaneously expressed by three subjects.

In the meantime, the selective memory impairment of LSD after long exposure has been used to form the basis of a new experiment. It was predicted, that schizophrenics would score significantly higher error on the FRT (immediate recall) provided a long exposure time was used. For a short exposure time, differences were predicted not to be significantly different. Both expectations were in fact borne out and results are ready for publication (5).

Judging by the differences between LSD and amytal in error scores (Figure 1 and t-ratios), the effect under discussion does not seem to be linearly related to exposure time, or the total amount of light stimulation. An optimum level of no significant difference between the drugs is indicated to lie near the one-second exposure time. A phenomenon similar in its paradoxical character at least has been obtained by Venables and Tizard (14) in schizophrenics. Here, reaction time, after initially decreasing with increasing light intensity, increased again at a specific level in contrast to normals. If no reasonable explanation can be offered, highly specific similarities between LSD and schizophrenia may now at least be studied for heterogeneous functions.

Of the numerous and already well-known introspections recorded during the experiment, the *subjective feeling of time* may be mentioned. Estimation of exposed lengths of time of up to 60 seconds, as well as their reproduction, was unsystematically tested but found to be accurate. However, the subjective feeling of time available for the inspection of the stimulus during short exposure times appeared greatly and consistently increased. The 1/100 sec. exposure, apart from rendering the stimulus "unbelievably clear and sharp", enabled S's "to see all the symbols (test shapes) individually". This is in marked contrast to the instantaneous perception in normals, or the rapid passing of time under amytal.

SUMMARY

Six normal subjects were given intravenous injections of lysergic acid and sodium amytal on separate occasions, followed by the recall version of the Figure Reconstruction Test. This test requires reproductions in drawing which are objectively scored for error and expressive movement. Using previous results obtained from schizophrenics and other patients, and equating the former with the action of LSD, the following predictions were made and verified. (1) Error of immediate memory is higher under LSD than under amytal. (2) Variability in expressive movement is greater for LSD than for amytal. (3) Spread of test shapes (expansion) is smaller for LSD than for amytal. Amongst other findings, a paradoxical effect was observed for LSD only, in that memory was worse after 15 seconds exposure as compared with a short time exposure of one second.

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D-AMPHETAMINE AND AMYTAL: I. EFFECTS ON MEMORY AND EXPRESSIVE MOVEMENT

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INTRODUCTION

"WE would predict that any test which has been shown to differentiate reliably and validly between introverts and extraverts will, when applied to subjects who have been administered an excitant (or depressant) drug, show shifts in scores in the direction characteristic of greater introversion (or extraversion)." "... the effects of depressant drugs are similar to those of brain damage as far as objective psychological tests are concerned. Conversely, the effects of excitant drugs are opposite to those of brain damage." These predictions by Eysenck (7) were tested using measures of immediate memory and expressive movement which had previously been validated against extraversion and brain damage. The measures discussed are derived from the Figure Reconstruction Test (immediate recall version).

Using Eysenck's equation of hysteria and brain damage with drug sedation the following predictions may be derived. Firstly, brain damage is known to be associated with *memory* defect. As far as the FRT is concerned, this has been demonstrated in that subjects with a known history of brain damage made considerably more errors than neurotics and non-neurotics (Bartholomew, 1). Furthermore, electroshock treatment significantly impaired memory (Bengelmann, 2). Persons to whom *amytal* is administered should therefore score *higher on error* than drug-stimulated persons.

On the other hand, it has been shown that, although not significantly so, a number of groups of extraverted subjects scored consistently lower on error than the introverts, using the learning version of the FRT (Bengelmann, 3). Secondly, employing the immediate recall version of the FRT, as used in the present experiment, extraverted male and female neurotics scored considerably lower on error than the introverts (Bengelmann, 6), provided a short exposure time was used. From this the prediction is made that *amytal decreases error*. These two opposing predictions show forcefully that Eysenck's theory on drug action is over-simplified.

Turning to *expressive movement*, as spontaneous or other acts accompanying involuntary movements, one would naturally expect sedatives to exert a calming effect. When referring to the literature, it has been pointed out that sedatives probably decrease movement variability and improve steadiness (Bengelmann, 5). As far as the presently used FRT is concerned it was found that hysterics, or "extraverted" neurotics, were significantly and consistently more variable in drawing than the dysthymics, or "introverted" neurotics (Bengelmann, 4). Furthermore, the present author has found that the application of electroshock leads to a considerable increase in variability on the FRT. These results are being prepared for publication. Consequently, Eysenck's association of hysteria with brain damage remains, in this respect, tenable.

The prediction to be made is that subjects should become more variable as the result of sedation.

Reference must be made at this point to one further test score, used in the present study, called *spread*, or the scatter of test items in drawings. Although logically similar to variability, this score is listed separately for various empirical reasons. It was previously shown that spread was consistently larger in hysterics, as compared with dysthymics (4). The prediction, correspondingly, would be that spread is highest for amytal.

Summarizing the predictions made from Eysenck's theory, disregarding the factual evidence, the following statement may be made. As a result of the administration of amytal, memory error, expressive movement variability, and spread scores will be significantly higher than after the administration of d-amphetamine.

METHOD

1. The Figure Reconstruction Test (immediate recall version)

As the principles of this technique have already been described, a short reference will suffice here (3, 4, 5). Five simple geometric shapes, distributed around a central circle, are exposed and drawn from memory (Fig. 1). *All* shapes must be reproduced. One test set consists of ten stimuli. Most of the scores are obtained with the help of the central ring, printed on the response sheets.

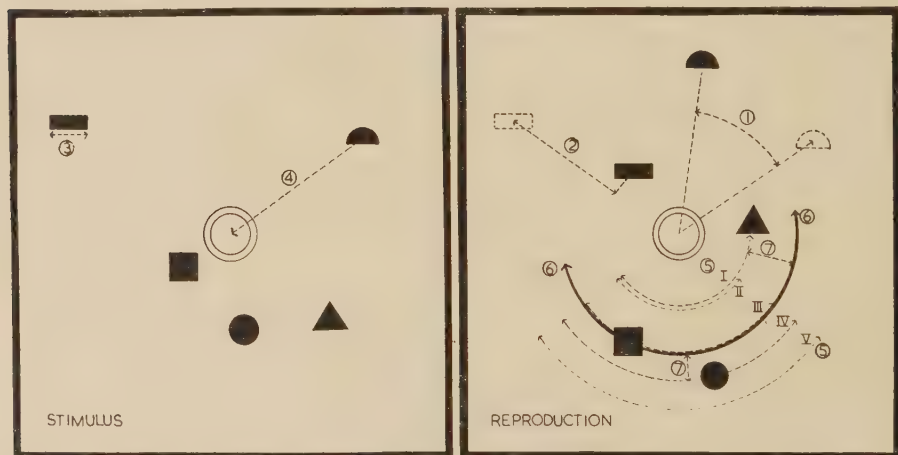


FIG. 1.—Example of stimulus card and moderately distorted reproduction. *Error scores:* 1. Rotation error. 2. Distance error. *Expressive movement scores:* 3. Size. 4. Distance. 5. Distance ranges I-V. 6. Mean distance (pattern mean). 7. Spread. Shapes drawn in dotted line in the reproduction indicate stimulus position.

In the present experiment a constant exposure time of one second was used, controlled by a compur shutter. The test cards were shown against a black background. A short exposure time was chosen in order to achieve a reasonable amount of error in the reproductions. Additionally, it is now known that an exposure time of this order favours the occurrence of differences between extraverts and introverts, whereas long exposure fails to do so (6). Unfortunately, some of the expressive movement characteristics, to be investigated, emerge more clearly under different exposure conditions. Variability scores appear to be favoured by very short and spread by long exposure time (5). As the same

subjects were tested by other experimenters there was not sufficient time to vary exposure duration. Nevertheless, results will show that a conclusion could be drawn despite adverse conditions.

2. Scores

Scores hitherto derived from the FRT are described in Figure 1 and the following:

Rotation error: mean axial displacement of the individual stimulus elements, measured in degrees (No. 1 in Fig. 1).

Distance error: radial, either centrifugal or centripetal displacement, measured in mms. (2).

Size: mean base length of reproduced shapes in mms. (3).

Size variability within shapes (SV within): mean variability of size between successive trials, scored separately for each category of shape (triangle, cross, etc.), in 1/10 mms.

Size variability between shapes (SV between): mean size deviations of the ten shapes from their mean, in 1/10 mms.

Distance: mean distance of shapes per trial from the centre of the reproduction sheet (6).

Distance variability between patterns (DV pattern): between trials variability of distance (6) in 1/10 mms. or, the mean deviation of a given number of distance scores from their mean.

Distance variability within ranges (DV ranges): identical with the preceding score, except for measuring separately at each of the five distances, or ranges (5, ranges I-V), at which S's choose to draw on the response sheet.

Distance spread: mean deviation of shapes from the mean distance within trials, as measured in mms. (7).

In addition to rotation and distance error, two more error scores were used. These are "*rotation proportion*", or the mean axial deviation in degrees of each reproduced shape from the *nearest* stimulus shape. (Rotation error, in contrast, measures same deviation of each reproduced shape from the *identical* stimulus shape.) Similarly, "*distance proportion*" measures the mean radial deviation in mms. of each reproduced shape from the *nearest* instead of identical stimulus shape.

Variability measures were used as ratios, as follows: SV within/size, SV between/size, DV pattern/distance and DV ranges/distance. Similarly, the distance error scores were, for each subject, divided by distance. It is obvious that the former depends heavily on the latter.

3. Subjects

There were six normal subjects, five men and one woman, all, with the exception of one technician, psychologists. The age range was 25 to 39, with a mean of about 30.

4. Design

The experiment was conducted concurrently with those reported by Eysenck and his co-workers (8, 9, 10, 11), using identical subjects and conditions. Drugs were given orally and consisted of 10 mg. d-amphetamine, $4\frac{1}{2}$ grains sodium amytal and a placebo. Tests took place on three different days using a balanced randomized block design to control for sequence effects. For greater detail the writer may refer to the above papers.

Before beginning the experiment, subjects were given three sets of tests in order to familiarize them with the task and to reduce practice effects. With three practice and three experimental tests a total of six parallel test sets had to be constructed. The experimental tests were systematically varied so as to avoid sequence effects.

RESULTS

1. *Immediate memory.* The mean scores were as follows:

			Amphetamine	Amytal	Placebo
Rotation error	..	..	37.4	41.2	39.7
Distance error	..	..	14.6	14.0	14.3
Rotation proportion	..	..	17.2	19.3	20.5
Distance proportion	..	..	7.4	6.8	7.1

As can be seen, means vary at random with treatments. Applying independent t-tests of related measures, the largest t-ratio between any pair of drugs was 0.790 (5 d.f.), which is not significant. It may be concluded that no significant differences were obtained relating systematically to drug treatments.

2. *Movement variabilities.* Results are shown in Figure 2, with the group means added in Table I.

TABLE I
Group Means Belonging to Figure 2

			D-amphetamine	Amytal	Placebo
Size variability (between)	..	..	4.43	3.78	4.13
Size variability (within)	..	..	2.43	2.45	2.57
Distance variability (pattern)	..	..	16.33	11.83	15.33
Distance variability (ranges)	..	..	11.83	10.83	11.67

Analysis of variance was used for all test scores. The between persons and between drugs variances were tested against the residual, as shown in Table II for distance variability (pattern).

TABLE II
Analysis of Variance of Distance Variability (Pattern) Score

Distance Variability (Pattern)				d.f.	s.sq.	m.sq.	F-ratio	Significant (Per cent.)
Drugs	..	..	..	2	67.000	33.500	4.855	5
Persons	..	..	..	5	132.500	26.500	3.841	5
Residual	..	..	..	10	69.000	6.900		
Total	..	..	..	17	268.500			

Total results of the analysis of variance are presented in Table III.

TABLE III
Results of Analysis of Variance Pertaining to Figure 2

				F-ratio	Drugs	Persons
Size variability (between)	..	..	..	4.922 (5%)	153.713 (0.1%)	
Size variability (within)	..	..	..	NS	18.021 (0.1%)	
Distance variability (pattern)	..	..	..	4.855 (5%)	3.841 (5%)	
Distance variability (ranges)	..	..	..	2.627 (NS)	5.017 (5%)	

D.f.: drugs 2/10, persons 5/10.

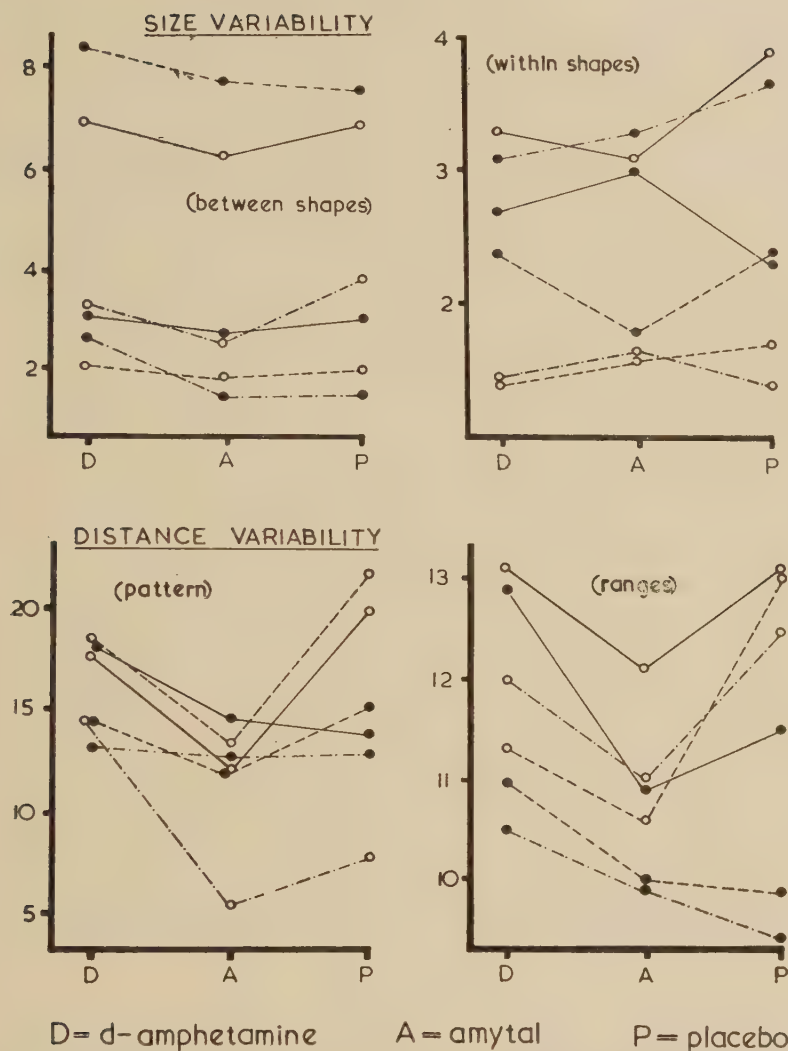


FIG. 2.—Four variability scores as related to drugs. Curves represent individual subjects.

As can be seen from Figure 2, all subjects scored higher with d-amphetamine than with amytal on three test scores, the remaining one being inconsistent in this respect. t-test analysis of the significant scores revealed differences between amphetamine and amytal to be significant at the 2 per cent level of confidence. The conclusion is drawn that amytal significantly decreased involuntary movement variability, as measured by the FRT. Additionally, it should be noted that the mean variability scores, with the exception of the least significant score, increased in the order amytal-placebo-amphetamine, indicating that opposing drug actions are indeed measured.

3. *Spread.* With an F-ratio of 6.066 (d.f. 5/10) differences between persons were significant (1 per cent.). As to drugs, the mean variance for the residual was higher than for drugs. The conclusion is that no significant differences

were found due to drugs. The mean scores were 22.8 (amphetamine), 24.3 (amytal) and 24.7 (placebo).

DISCUSSION

The results on *memory* do not support the predictions made from Eysenck's theory, although an exposure time favourable to the production of differences between extraverts and introverts was chosen. As LSD was previously shown to produce considerably higher rotation error than amytal (5), a comparison with the presently used stimulant may be of interest. The suggestion may be derived that differences in drug action between LSD and d-amphetamine may be measured with a memory test. Results regarding *variability* were, in agreement with the literature, found to be contradictory to Eysenck's theory. As to *spread*, again, no support can be drawn for the prediction made. In this case it may be argued that a longer exposure time was needed for significant differences to occur.

The criticism may be made that predictions for the test scores used cannot be derived from Eysenck's theory of extraversion. This, however, is of little importance as predictions could be made on the basis of empirical results, which is obviously superior to speculation.

A joint consideration of the experiments performed by Eysenck and his co-workers (8, 9, 10, 11) and of those by the present author suggests that, at present, the results obtained with amytal and amphetamine are better understood on the basis of the implied pharmacological actions than from personality theory point of view. Eysenck, in his introductory paper on methodology on drug research (7) has apparently relied too heavily on Shagass' (12) reports that "sedation threshold" discriminates neurotic categories as expected from Eysenck's theory of extraversion. The high reliability, quoted by Shagass, appears suspect in view of a recent study directed to this point. Thorpe and Barker (13), copying Shagass' technique, asked 16 psychiatrists to note the point, in tape records, at which slurred speech definitely occurred. The conclusion was that "speech slurring is too subjective to be of general use, and that some doubt is thrown on the objectivity of the Sedation Threshold".

SUMMARY

Eysenck's theory that the effects of drug sedation are those of increased extraversion, whereas stimulant drugs decrease scores related to this factor, was tested using memory and expressive movement scores of relevant validity. On memory scores, neither significant nor consistent differences were found between amytal and amphetamine. Movement variability was reliably higher with amphetamine than with amytal. Findings were interpreted as either not supporting or as contradictory to Eysenck's theory of drug action.

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D-AMPHETAMINE AND AMYTAL:

II. EFFECTS ON CERTAINTY AND ADEQUACY OF CERTAINTY IN RECALL AND RECOGNITION

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INTRODUCTION

IN the present experiment measures of certainty and adequacy of certainty, derived from a test of immediate recall and recognition, were investigated in relation to d-amphetamine and amytal.

There is *clinical evidence* that these drugs have specific effects on intra-personal feelings of certainty and adequacy. Amphetamine is known to increase "self-confidence" and "hope with determination not to give in" (9), or to increase "ego strength and self-esteem" (11). Sodium amytal may lead to a reduction in "critical appraisal of perceptual clues" and "apprehensive caution with which plans of action are usually integrated to such perceptions" (6). Mûcher (7) has provided experimental evidence that diethylbarbituric acid produces "increased sense of frustration and threat to adequacy-feeling". "Intellectual control" was found to be reduced.

The repeated attention which certainty has attracted in experimental situations (8), the bearing of judgment in level of aspiration on personality traits (3) and the recent emergence of reliable response sets in paper-and-pencil tests (10) have occasioned the need for more detailed analysis and the construction of simple and objective research tools. Outside the field of paper-and-pencil tests, apparatus measuring certainty has varied according to special circumstances. No objective techniques, easily scored and applied in both individual and group sessions, appear available.

The *first concern* of the present experiment is then to validate such a tool, using a stimulant and a depressant drug as suitable means to vary subjective states of certainty. The *second aim* is to gain insight into the relationship of certainty to two test conditions, immediate recall and recognition of visual material. Although it is known that a variety of different tests are responded to with fairly highly correlated certainty, it has also been found that notable and systematic exceptions occur (5). Some of the reasons for this may be sought in variations of insight in the correctness of performance, of difficulty of task and of psychological function investigated. The test used in the present study is designed to favour the appearance of differential results by employing two conditions of memory, recall and recognition, which are likely to vary considerably in their level of difficulty.

From Eysenck's (3) level of aspiration experiments, it can be seen that judgment of positive confidence in test performance is significantly higher in (extraverted) hysterics than in (introverted) dysthymics. As more recently (4) extraversion has been linked with the action of depressant drugs, for example amytal, and introversion with stimulant drugs, for example amphetamine, the *first prediction* is that certainty increases with amytal relative to amphetamine. As the judgment of performance in level of aspiration tasks was more correct,

or more adequate, in hysterics than in dysthymics the *second prediction* is made that amytal increases adequacy of certainty relative to amphetamine.

These predictions appear contradictory to clinical experience, quoted above. With raised "self-confidence" and "self-esteem", as observed after amphetamine, an increase in certainty may be expected. Similarly, "reduction in critical appraisal" and "threat to adequacy-feeling", as seen after the application of amytal, may parallel reduced adequacy of certainty, as measured in the present experiment.

METHOD

1. *The Figure Reconstruction Test (immediate recall and recognition)*

This test, referred to as FRT, requires drawn reproductions of visually presented patterns containing five simple geometrical shapes. One test set of the immediate recall version used in the present study consists of ten patterns. They are exposed for half a second, followed by reproduction after each single exposure, as described in Part I of the present series of papers (2). After S has completed the recall portion of the test, the ten patterns are mixed up at random with 20 additional patterns, similar in character but unknown to S. The order is constant for each set of 30 patterns but varies slightly between the parallel sets used. During recognition S is required to make two statements after each exposure of five seconds duration, *firstly* whether he thinks he has seen the pattern during the recall period or not and, *secondly*, how certain he feels about his positive or negative response.

The following *grades of certainty* are used:

- +2=very certain
- +1=somewhat certain
- 1=somewhat uncertain
- 2=very uncertain

During *recall* S's were required to add one of the above grades in writing to each of the five shapes reproduced in a pattern. Fifty judgments are therefore obtained for ten patterns. +2 means: "I am very certain that this shape is correctly reproduced." During *recognition* one of the above grades is attributed to each of the 30 patterns presented (total of 30 judgments). +2 here means: "I am very certain that I saw this pattern during the recall period."

2. *Scores*

The following scores were used.

Degree of certainty (recall): mean degree of certainty per shape, regarding signs, range +2 to -2 expressed in graphs as +200 to -200.

Degree of certainty (recognition): mean degree of certainty per pattern, otherwise as above.

Adequacy of certainty (recall): mean error score for all uncertain responses (-1 and -2) minus mean error score for all certain responses (+1 and +2). Results are reported separately for four error scores, described in Part I of the series: rotation error, rotation proportion, distance error and distance proportion. A large difference score is taken to indicate adequate behaviour. A small, or negative score means inadequate attribution of certainty to performance.

Adequacy of certainty (recognition): scored as shown by the following example (recognition responses total 30 per test).

						Sum certainty	
Number of positive responses (subjective recognition):						11	
of these: correct						6	+5
wrong						5	-2
Number of negative responses (subjectively not recognized):						19	
of these: correct						15	+6
wrong						4	+4
Certainty for correct responses							+11
Certainty for wrong responses							+2
Score							+9

To respond "adequately" statements of certainty must be positive for correct and negative for wrong recognition. In such a case the sum certainty scores for both correct and wrong responses are added. If, as in the above example, certainty is (adequately) positive for correct answers, but also (inadequately) positive for wrong answers, the latter score is subtracted from the former. The total score is then $11 - 2 = 9$.

3. Subjects, Drugs and Design

Six normals participated in the experiment. Drugs were given orally and consisted of 10 mg. d-amphetamine, $4\frac{1}{2}$ grains sodium amytal and a placebo. Tests took place on three different days, using a balanced randomized block design to control for sequence effects. A full introductory test was given before the experiment. Parallel test forms were constructed for each session and varied in a balanced order. More information is provided in the first paper of the series.

RESULTS

Results were treated by means of analysis of variance. The "between persons" and "between drugs" variances were tested against the residual, as shown in Part I. Additionally, some t-tests were carried out. The main results for degree and adequacy of certainty are shown in Figures 1 and 2. Scores, from which graphs were constructed, are given in Table I.

TABLE I
This Table Shows Scores Pertaining to Figures 1 and 2

				Degree of Certainty					
				Recall			Recognition		
Subjects			D	A	P		D	A	P
1			26	42	46		70	73	80
2			40	44	54		47	10	97
3			-24	12	52		-10	-27	13
4			52	64	38		80	77	93
5			30	64	22		17	27	20
6			56	108	54		40	50	87
Mean	..	..	30.0	55.7	44.3		40.7	35.0	65.0

TABLE I—continued

Group means negative minus positive certainty	Adequacy of Certainty			Recognition		
	Rotation Error:	Rotation Proportion:	Distance Error:	Subjects	11	10
	33.3 27.0 30.5			1	6	3
	9.3 4.2 10.0			2	—6	16
	7.8 5.8 6.6			3	13	22
	6.7 —4.7 6.0			4	20	6
				5	7	2
				6	9	9.8
				Mean ..	9.0	1.0

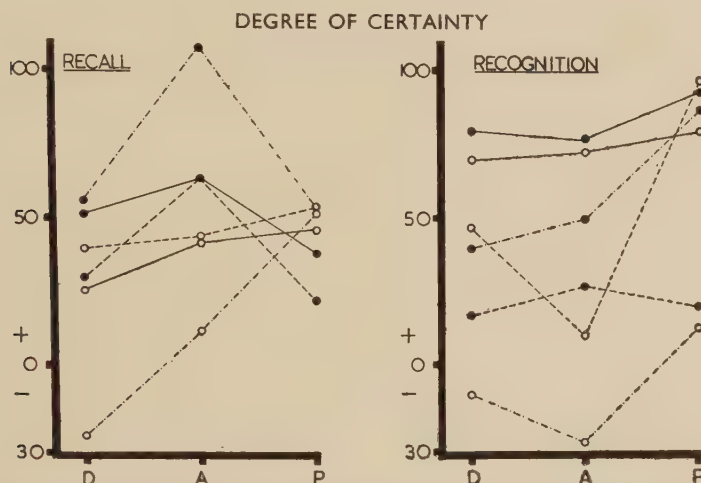


FIG. 1.—Inconsistent results with degree of certainty. In recall (easy task) drugs affect certainty specifically, amytal scoring highest in all six subjects. In recognition (difficult task) both drugs lower certainty.

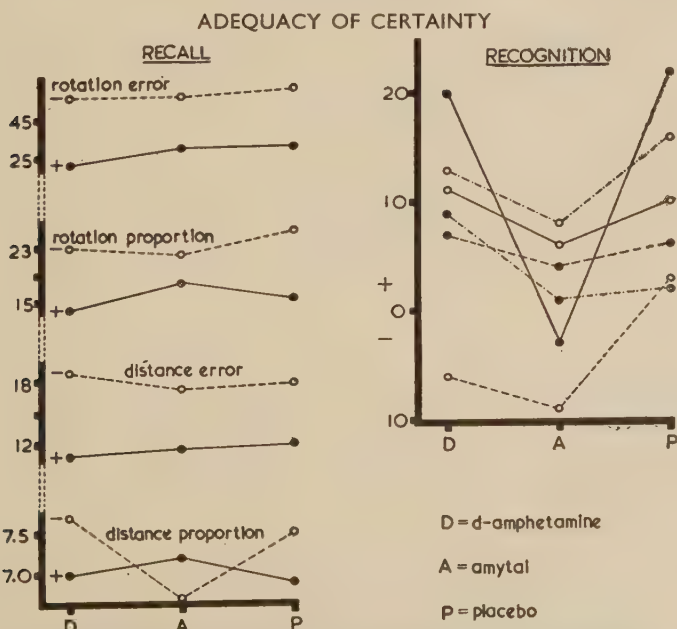


FIG. 2.—Recall: The difference between mean error for uncertain (—) and certain (+) responses is lowest for amytal (most inadequate).

Recognition: Adequacy of certainty is for all six subjects lowest after amytal.

It is pointed out that results obtained may be considered independent of corresponding recall or recognition performance. In Part I neither significant nor consistent differences between drugs were found for the various error scores used (2). In the present study correctness of recognition was as follows:

Mean Correct Recognition	D-amphetamine	Amytal	Placebo
10 patterns seen during recall	5.6	5.2	5.8
20 patterns not seen during recall	12.8	12.2	12.4

For both portions of recognition the means are very similar between the drugs. Only slightly more than half of the patterns were correctly recognized. Analysis of variance revealed differences between drugs to be highly insignificant.

1. *Degree of certainty in recall.* From Figure 1 and Table I it can be seen that degree of certainty is highest for amytal and lowest for amphetamine. Results are consistent for all S's, the difference between these two drugs being significant at the 2 per cent. level of confidence ($t=3.49$, 5 d.f. one tail test). The first prediction derived from Eysenck's drug theory appears confirmed for the recall part of the test, although it must be stated that the overall significance of these results is neither significant for drugs ($F=2.24$, d.f. 2/10) nor for persons ($F=2.54$, d.f. 5/10).

2. *Degree of certainty in recognition.* Results obtained from the analysis of variance were significant with an F-ratio of 5.09 (5 per cent.) for drugs and one of 11.80 (0.1 per cent.) for persons. They are, however, distinctly different from those reported in the preceding paragraph in that degree of certainty is highest for placebo and lowest for amytal. The first prediction is therefore not confirmed for the recognition part of the test. No significant differences were obtained between the experimental drugs. Differences were greatest between amytal and placebo ($t=3.00$, 5 d.f., significant at the 5 per cent. level) and between amphetamine and placebo ($t=2.44$, 5 d.f., 10 per cent. level). These results suggest that drugs acted unspecifically by lowering certainty in a similar manner against the placebo effect.

3. *Adequacy of certainty in recall.* Results show responses to be most inadequate after amytal for all four error scores (Fig. 2, Table I). In the case of "distance proportion" error for certain responses was even *higher* than for uncertain responses, after amytal had been given. None of the corresponding F-ratios was, however, found to be significant, as shown below.

Adequacy in Recall				Drugs (d.f. 2/10)	Persons (d.f. 5/10)
Rotation error*	..	..	..	—	—
Rotation proportion	..	..	..	1.85 (NS)	1.32 (NS)
Distance error	..	..	..	2.04 (NS)	3.00 (NS)
Distance proportion	..	..	..	2.09 (NS)	3.20 (NS)

* Residual variance larger than "between" variance.

A further analysis revealed that, with the exception of rotation error, t-ratios between amphetamine and amytal varied between the 10 and 5 per cent. levels of significance. Furthermore, for six S's and four error scores responses for amytal were 19 times more inadequate, once equal to and four times more adequate than for amphetamine. In all, it may be suggested that

responses tended to be more inadequate for amytal than for amphetamine. Further support for this is given in the following paragraph.

4. *Adequacy of certainty in recognition.* Responses made under amytal were, without exception, more inadequate than those made following either amphetamine or placebo (Fig. 2, Table I). Analysis of variance revealed no significant differences. Using Fisher's sign test, however, comparisons between amytal and any of the two remaining conditions may be considered significant at the 2 per cent. level of confidence.

Considering the overall consistency of results "within" and in particular "between" the two experimental conditions, recall and recognition, there is strong support for the suggestion that performance was more inadequate for amytal than for amphetamine. This contradicts the second prediction derived from Eysenck's drug theory.

DISCUSSION

A joint consideration of results on *degree of certainty* shows that Eysenck's drug theory, equating sedation with extraversion and stimulation with introversion, is not confirmed. The differential response of amytal to recall and recognition, deserves further clarification. As to *adequacy of certainty* results consistently contradicted Eysenck's theory (prediction 2). When clinical observation, discussed in the introduction, is considered results are equally inconsistent for degree of certainty, but fitting for adequacy of certainty, with the following reservation. As regards adequacy, amphetamine and placebo acted in a similar manner, unlike amytal. This finding agrees with the well-known fact that stimulant drugs of this kind in general do not improve performance unless it is impaired during the time of administration.

Differences in degree of certainty, as found between recall and recognition, may be due to corresponding differences in levels of difficulty. In recall, mean rotation error was found to be near 40 degrees. As chance performance on the FRT is 90 degrees (1), the task level used here may be considered as fairly easy. In recognition, as was stated above, correct performance was only little above chance level. Recognition may, therefore, be considered to have been much more difficult than recall. This would be consistent with S's subjective impression, who invariably considered recognition the more difficult task. The working hypothesis may be derived that difficulty level forms an important variable and that both stimulant and depressant drugs alike depress "certainty" feelings in the face of high levels of difficulty. Drug specific action may then increase with easiness of task.

SUMMARY

Two predictions, derived from Eysenck's theory on the action of stimulant and depressant drugs, were tested. The predictions stated that both degree and adequacy of certainty judgments, obtained from recall and recognition tasks, would increase with amytal and decrease with amphetamine. Eysenck's theory was either not supported or contradicted. For degree of certainty, results were inconsistent between recall and recognition which was interpreted as due to differences in levels of difficulty. As regards adequacy of certainty, scores were consistently lowest for amytal.

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THE "INEDUCABLE" CHILD

By

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A SMALL number of children have brains which are so malformed or damaged that they make little response to special methods of education. A survey of the forms of brain abnormality in these cases has been carried out by Crome (1954). The category of children who can properly be classed as ineducable is, however, strictly limited and should, probably, be confined to idiots. Williams (1957) has pointed out that the task carried out in occupation centres for imbeciles is that of "educating the ineducable", whilst a number of recent studies have shown that imbeciles have much more educational capacity than is generally assumed.

My concern here is with children who are above imbecile level but who have been placed in institutions for mental defectives or excluded from school as ineducable under section 57(3) of the Education Act of 1944. This exclusion is carried out on the basis of a medical report completed by the medical officer of the local authority, though the Act does make provision for consideration by the Education Authority of "any reports or information which the local education authority are able to obtain from teachers or other persons with respect to the ability and aptitude of the child".

Prior to the passing of the 1944 Education Act the term "feeble-minded" in regard to children was applied to those who "appeared to be permanently incapable by reason of such defectiveness of receiving proper benefit from the instruction in ordinary schools". Since that time children attending schools for the educationally sub-normal have not been certified as mentally defective and the term "feeble-minded" has covered a much more limited category of children defined by the Mental Deficiency Act as suffering from "disability of mind of such a nature and extent as to make them, for the purpose of section fifty-seven of the Education Act, 1944, incapable of receiving education at school". According to section 57(4) of the Education Act a child is incapable of receiving education at school "not only if the nature and extent of his disability are such as to make him incapable of receiving education, but also if they are such as to make it inexpedient that he should be educated in association with other children either in his own interests or in theirs".

A child is "subject to be dealt with", i.e. to be placed in an institution or under guardianship under section 2 of the Mental Deficiency Act if he is "for the time being subject of a report in force under the enactments relating to education that he has been found incapable of receiving education at school, or that by reason of a disability of mind he may require supervision after leaving school". The words "for the time being" were inserted in 1948 by the Education (Miscellaneous Provisions Act) and represent a shift away from the old notion of permanent incapacity.

O'Connor and Tizard (1954) found that 12 per cent. of patients in mental deficiency hospitals in the home counties were children. This was the same figure as that given by the Ministry of Health in the Report for 1949. Taking the 1953 figure of some 57,000 mental defectives in hospitals in England and

Wales, 12 per cent. of this gives 6,840 children in hospitals, whilst the local authorities have some 17,000 child mental defectives in their care, making a total of 23,840 children excluded from the education service. Unfortunately the Medical Research Council survey undertaken by O'Connor and Tizard referred to above did not include any assessment of the intelligence of the children, but 11 out of 73 in their sample were classed as feeble-minded. The statutory classification was used as a basis here and it has been found in our experience at the Fountain Hospital that this is of limited value. Although, as pointed out above, the term "feeble-minded" should be reserved for those whom it is "inexpedient" to have in a class with others, very often the certifying medical officer will use it for cases about whom he has some doubt, or possibly because he does not like to take the step of labelling a very young child as an idiot or imbecile. On the other hand some children who are above imbecile level show up badly on ascertainment and are underestimated. Others improve in their attainments as they grow older. It is generally agreed that any assessment of intelligence prior to the age of five years is an unreliable guide to future educability.

Ages of ascertainment of children as mentally defective, or as ineducable or of admission to mental deficiency hospitals are not published. In the Medical Research Council survey 11 of the 73 children considered were under the age of five years at the time of the survey and it is probable that a considerable proportion of the others were also under five at the time of their admission to hospital and hence had no trial at school.

If we take the figure of 11 feeble-minded among 73 children in the M.R.C. sample as a guide there might be something like 3,592 children regarded as of feeble-minded level amongst those excluded from school, there being no reason to suppose that those in the care of the local authorities are of lower grade than those in hospital. However, for the reasons mentioned above such a formal classification is of limited value.

It may therefore be of interest to consider the mental level of a series of children admitted to hospital as mentally defective and the proportion among them who were found to be above imbecile level.

143 ADMISSIONS TO FOUNTAIN HOSPITAL

All children admitted to the hospital in the two years prior to 30 June, 1955 were considered. They were admitted primarily from the counties of London and Surrey. Temporary admissions were not taken into account. The average age on admission was 3.75 years (standard deviation 2.1 years, range 6 months to 12 years 8 months). Information about these children was available from direct psychiatric examination, from the nursing staff about their behaviour in the ward and from the occupation centre in those instances where they were of sufficient level to attend. In practice all those who can walk and make some attempt to co-operate in the simplest group and individual activities do attend whilst some separate provision is made for cripples. Patients are also routinely examined by the psychologist who compiles a report which includes the result of any formal test found applicable. In many cases it was found only possible to score the child on a "Social Maturity Scale". This applied particularly to the first interview. On subsequent occasions it was often possible to give some other test.

On scrutinizing all the information available about each child it soon becomes clear that there is no simple method of deciding whether he is above

imbecile level, still less whether he should be recommended for reconsideration as educable. Quite a number of the children had multiple handicaps such as deafness, blindness, cerebral palsy, specific speech defects, and epilepsy, which needed to be taken into account in assessing the results of psychological examination as well as in taking a decision as to acceptability for schooling. In addition some of the children had special psychological problems ranging from psychosis to some difficulty in adjustment to the hospital environment. A further problem, which appeared to be particularly important in the case of the more intelligent children and which is the subject of a separate communication (Craib and Woodward, 1958), is disturbance or inadequacy in the home environment. This factor is operative prior to admission, to some extent during the stay in hospital and also reflects on the chances of subsequent successful adjustment within the educational system.

The value of psychological assessment of educability was limited by the very low age of many of the patients and by the fact that in the case of the younger and more retarded patients it was only possible to apply a developmental scale. There was considerable variation between the results of such a scale and those of other types of test. There was often some discrepancy between the results of verbal and non-verbal tests. In some cases there was quite a marked change in the results scored when the test was repeated after an interval; any results obtained on children soon after admission to hospital are considered to be of limited value as a guide to educability.

When the results obtained by these 143 children were listed it was seen that 33 (23 per cent.) scored a social quotient or intelligence quotient of 50 or over on some test at some time. It seemed, however, that only fourteen of these were likely to receive serious consideration as candidates for special school. One of the main reasons for this curtailment of the list is that whereas a score of 50 or more on the Vineland scale indicates a certain level of social adjustment a child is unlikely to be accepted into school unless there is a minimum of verbal development. It is possible that some of the younger children in this series will become more verbally adequate and may prove to be educable, though institutional conditions are not favourable to verbal development.

The better verbal level of the fourteen children who were looked upon as possible candidates for school is shown by the fact that 13 of them achieved an intelligence quotient of 50 or more on a test of the Binet type (Stanford-Binet for the sighted and Langan revision for the blind). The results of the scrutiny of this sub-group of 14 from the standpoint of educability are set out in Table I.

Most of these children seemed to be of limited intelligence so that it was more difficult for them to overcome additional handicaps than for children of greater intellectual capacity. On the other hand the existence of an additional handicap may create the impression of an intellectual capacity lower than that which actually exists. The "social quotients" of three of the blind children mentioned above were 35, 32 and 48, i.e. very much lower than the Langan scores. Unless such blind children receive a good deal of special attention they are liable to fail in the various social accomplishments which are scored on such a scale.

It will be seen from consideration of this group that the term "ineducable" has a very arbitrary meaning. In practice much depends upon the education authorities. Policy is likely to be influenced by the availability of residential and day accommodation in schools for the educationally sub-normal, by the criteria for admission laid down by the teachers in those and other schools and

TABLE I

Successful:	Number	Stanford-Binet I.Q.
In special school	3	67, 68, 52
(One subsequently excluded because of psychopathy)		
In deaf unit prior to school	1	86, —104 (Merrill-Palmer)
On waiting list for blind school	1	64*
Unsuccessful:		
Recommended for special schooling but rejected ..	1	55
(Rather rigid and aloof, did not co-operate with school medical officer)		
Psychotic	1	51
Epilepsy and multiple handicaps	1	64
Speech defect and multiple handicaps	1	61
Marked hydrocephalus	1	60
Blind	3	54, 55, 64*
(One attending occupation centre after discharge from hospital)		
Borderline imbecile	1	

*Results of Langan Adaptation.

by the manner in which regulations and policy are interpreted by the school medical officers carrying out statutory examinations. It is clear that in any event there is considerable overlap in intellectual ability or test performance between the group of children who are in mental deficiency institutions and the group in special schools.

DISCHARGE OF CHILDREN TO E.S.N. SCHOOLS

It would appear that many large institutions catering for mentally defective children seldom return such children to the educational system even if they find them to be appreciably above imbecile level. This situation may be partly ascribable to the inertia in the administrative apparatus and the considerable amount of paper work and legal formality which is necessary in order to secure a reversal of a decision as to ineducability, to discharge a child from the mental Deficiency Acts and to find a suitable place for him in school. Another consideration which is important is the view commonly held that the occupation centre within the mental deficiency hospital is in effect a school. This view is reinforced in those cases where the centre is staffed by qualified teachers. Those who support this view hold that the child can receive education appropriate to his needs within the hospital.

In practice in our hospital we have taken the opposite view on three accounts. Firstly, children who are brought up in institutions tend to lag in mental and emotional development owing to lack of suitable stimulation, limited experience, monotony, routine, lack of opportunity for individual expression, too protective or too restrictive a regime and absence of opportunity for forming emotional relationships within a more limited group.

A second problem concerns the educational level attainable within an institution. A survey of the type of staff employed in occupation centres within mental deficiency hospitals shows great diversity. Nurses, craftsmen, teachers of the mentally handicapped (occupation supervisors who have completed a course organized by the National Association for Mental Health), occupation therapists, schoolteachers and others are engaged in this work. Owing to the shortage of suitably qualified staff there are also many staff with no specific qualifications. The impression is that whereas the staff of hospital occupation

centres well appreciate and understand the limitations of the children in their care they do not always fully realize and take advantage of the capacity of certain of the children for further educational development. The very location of the centre within the hospital may also have an important bearing on the results achieved. It is noteworthy that in a comparable field O'Connor (1953) found that output of defectives on a particular job was improved greatly if the job was done in a factory rather than in hospital.

Finally, it is a serious disadvantage at the present time to be stigmatized as mentally defective and as needing detention within a mental deficiency hospital. A person with this history is likely to find more difficulty in occupational placement and in gaining social acceptance. It is desirable therefore that this should be avoided when possible.

With these considerations in mind we have during the past 8 years and with the co-operation of the appropriate local authority, public health and educational services made vigorous efforts to reinstate children who seemed likely to be acceptable within the educational framework. A series of 44 such children who have been accepted as educable is considered by Craib and Woodward. A preliminary survey was carried out by Mrs. Beatrice Fliess-Hermelin on 13 of these children, some time after their discharge from hospital and from the Mental Deficiency Acts, to ascertain the extent of their educational progress in school and adaptation to conditions of normal life in the community. Permission was requested from the Local Authority to examine a group of 25 children, of these, however, four had been rejected at the time of the survey and arrangements for the examination of the others were not completed in time for their development to be studied. In order to provide some sort of basis for comparison it was thought useful to examine the progress over a similar period of time made by a group of 12 children above imbecile level who for various reasons had not been placed in school, and had remained in the institution. The mean Stanford-Binet intelligence quotient of the group remaining in the hospital was 54.5 (range 42-64) whilst that of the group placed in school, prior to discharge was 65.3 (range 56-75). The two groups were not strictly comparable, both on account of the difference in intellectual ability and because of other factors operative in the selection of the group to be placed in school.

In considering the group of children who remained in hospital (Table II)

TABLE II
Children Remaining in Hospital

Patient	Date of Birth	I.Q.	Chronological Age		Mental Age		Graded Vocabulary		Comprehension		Arithmetic		Drawing Score	
			y	m	y	m	y	m	y	m	y	m	y	m
A. Girls:														
1 ..	26 5 45	58	10	3	5	11	f		f		f		7	3
2 ..	13 5 42	57	13	3	7	6	f		f		f		7	9
3 ..	11 4 46	56	9	4	5	3	f		f		f		4	3
4 ..	29 6 47	58	8	4	4	10	f		f		f		4	4
5 ..	13 5 36	51	19	3	7	7	f		f		f		9	6
6 ..	7 3 40	52	15	5	7	7	4	0	f		f		7	9
B. Boys:														
7 ..	10 9 37	42	17	11	6	4	5	6	5	5	f		6	0
8 ..	18 3 36	57	19	5	8	7	6	0	6	0	6	10	10	10
9 ..	23 5 37	64	18	3	9	8	5	6	6	5	6	10	8	9
10 ..	31 1 40	51	15	7	7	7	f		f		f		8	0
11 ..	6 6 39	51	16	2	7	7	f		f		6	2	10	0
12 ..	12 7 37	59	18	1	8	11	5	6	6	0	6	10	9	6
Average ..		54.5	15											

f—failed to pass easiest item on test.

it will be noted that the educational attainment of the boys seemed appreciably better than that of the girls. The average age of the boys was $17\frac{1}{2}$ compared with $12\frac{1}{2}$ for the girls which probably accounts for the greater part of the difference: in addition to this the boys in question have the advantage of being at Hastings in a smaller unit which is an annexe of the general hospital, of a wider range of occupations and more contact with people outside hospital.

If the assessments of intelligence quotient obtained before and after discharge from hospital are assumed to be comparable, then the children placed in special schools have changed little in this respect (Table III). The 1955 ratings on these

TABLE III
Children Discharged to E.S.N. Schools

Rating in 1955																
Patient	Date of Birth		I.Q. Before Discharge	I.Q.	Chronological Age		Mental Age		Graded Vocabulary		Comprehension		Arithmetic		Drawing Score	
					y	m	y	m	y	m	y	m	y	m	y	m
1	..	4 9 46	68	61	9	0	5	6					6	2	6	9
2	..	4 2 45	57	49	10	7	5	5			f		f		6	0
3	..	8 9 40	70	80	15	0	11	6	11	6	11	0	11	0	13	6*
4	..	10 9 45	?	(57)	10	0	5	8	6	0	6	0	f		—	
5	..	20 10 41	70	66	13	11	9	0	6	6	6	0	6	5	9	9
6	..	1 5 44	59	55	11	4	6	3	7	0	6	2	6	2	6	9
7	..	12 11 45	75-80	70-80	9	10	7	6	6	0	5	2	6	2	8	6
8	..	6 9 39	60	67	16	0	10	0	11	0	8	0	8	6	10	6
9	..	1 8 43	65	50	12	1	6	2	f		f		6	2	6	0
10	..	3 3 42	73	78	13	6	10	4	13	0	7	0	7	6	9	9
11	..	7 8 39	56	52	16	1	7	10	9	0	8	0	6	6	8	6
12	..	13 2 41	58	47	15	7	6	8	5	6	5	0	6	4	9	6
13	..	10 7 39	65	70	16	2	10	6	7	6	7	6	7	3	13	6*
Average			64.9	62.5	13	5										
f—failed easiest item in test.																
*—maximum score.																

f—failed easiest item in test.

*—maximum score.

children were all carried out by Mrs. Beatrice Fliess-Hermelin whilst the earlier assessments were done by one of four psychologists.

The educational assessments of these school children surpass or equal the mental age in 6 cases. One child was difficult to test on account of gross motor handicap. It was thought that cases 3 and 10 who, although having a relatively good intelligence level, had done badly on tests of educational performance, were handicapped by the low level of the class in which they were, whilst in two other cases with less satisfactory results, emotional factors were thought to be important. In general, however, these results clearly demonstrate that the term "ineducable" is not applicable to this group of children. They also reinforce the need for great caution in using this term. The group sent to E.S.N. schools overlaps with some members of the group used for comparison and it seems reasonable to suppose that if this group had had similar opportunities their educational level might have been much higher.

There are wide differences between different schools and even wider differences between different institutions providing for backward children. Much will depend, for example, on the size of the unit and on the rigidity or otherwise of the regime. The impression gained from the present survey was that the advantage of the school for the children studied was twofold. On the one hand they have the benefit of a somewhat more formal education, on the other they are treated essentially as children to be educated and assisted to develop. In hospital, there is always a danger that the child will be treated basically as a patient. There is thus a tendency to protect and help him. This difficulty is well recognized to exist, being particularly pronounced when institutions are overcrowded and understaffed.

Of recent years an arrangement has been arrived at between this hospital and the local authorities concerned, that children admitted to hospital who subsequently appear to be educable may be sent to school on a trial basis. In some cases they go for resident trial, in others on a daily basis. Trial may last 6 months or a year. This arrangement has proved extremely satisfactory and indeed seems to be the only logical method of assessing educability in a borderline or debatable case. It is recommended for wider adoption.

As a longer term measure it is hoped that the proposed revision of the mental deficiency legislation will improve the position of this group of children. In particular the transfer of the Occupation Centres to the education authorities, the abolition of the term "ineducable" and the introduction of a voluntary system of placement for mentally defective children without formal "certification" or "authority for detention", as in other hospitals, is recommended. It is suggested that in this way it would become much easier to obtain for children an appropriate form of education and training at any age and that an early decision on this matter would not prejudice a subsequent review of each child's needs.

SUMMARY

The term "ineducable" should be abolished or confined to a very limited number of idiot children. Imbeciles have more educational capacity than is generally assumed. A number of children classed as ineducable are above imbecile level. Some of these are still classed as "feeble-minded" in the pre-1944 sense of the term. On the basis of the Medical Research Council survey there may be some 3,592 children classed as feeble-minded but this classification has very limited value. Of 143 consecutive admissions to the Fountain Hospital, 14 (9.8 per cent.) were above imbecile level. Of these 6 were recommended for education in school. Five were accepted by the education authorities and one was subsequently excluded. Seven of the remaining 8 children had additional disabilities. The results of intellectual assessment of the children varied on different tests and at different ages.

The institution environment is unsuitable for children above imbecile level because (a) it stigmatizes the child in later life, (b) character development may be inhibited, (c) formal schooling is not usually available. Forty-six children have been discharged to school in the past 8 years from the Fountain Hospital. The intellectual level of 13 of these children after some years in school was studied as was that of 12 children above imbecile level remaining in hospital. The results on intelligence tests of those discharged had not improved but they were relatively much less retarded academically than those remaining in hospital. In 6 cases educational assessment surpassed or equalled mental age among those discharged. The results show that these children were not "ineducable". The arrangement of informal trial for 6-12 months in special schools for such "borderline" children is recommended. Under new legislation it is suggested that occupation centres be transferred to the education authority and that informal admission to hospital should replace "certification".

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MENTAL ABNORMALITY AND MILITARY DELINQUENCY

By

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DURING World War II considerable attention was given by the War Office to problems of delinquency in the Army and a number of psychiatric investigations were carried out with the ultimate aim of reducing the rate of military delinquency. Some of this work was specifically concerned with the relationship between mental abnormality (mental disorder and defect) and military delinquency. The purpose of this paper is to survey the unpublished reports, together with the published literature, and point to general conclusions.

Evaluation of the connection between mental abnormality and military delinquency is made difficult owing to the same problems met by those who made the original investigations. Factors such as fluctuation in diagnostic rates due to changes in administrative policy, or the complication of differential diagnosis by malingering and other forensic issues, must make generalization from these studies rather tentative. Nevertheless, the consistency of the information suggests that it may be used as a reliable basis for inference.

During 1945, 450 soldiers awaiting court-martial in one Command were referred for psychiatric examination. Thirty per cent. of these were considered to be "totally unfit for any form of military service" and another 50 per cent. showed evidence of "some intellectual or temperamental disability" (Mackenzie, 1946). As only one-seventh of all those awaiting trial were examined, the figures suggest that some disability might also have been found among many of the others had they too been referred for examination.

In a psychiatric investigation of a Young Soldiers' Battalion with particularly numerous disciplinary problems, Kennedy (1941) found that 25 per cent. of the discontented men showed marked psychiatric disability. This rate was much higher than among offenders in comparable units. Kennedy decided that most of the delinquency resulted from low morale brought about by poor administration and the bad methods used to train these immature soldiers. Situational factors had precipitated breakdown, and the most severe reactions were produced by those who were seriously predisposed.

An estimate made in 1942 was that fully one-third of all soldiers in detention suffered from psychiatric disabilities, the most common being mental deficiency (AG 1, 1942). At that stage of the war the system of scientific personnel selection was only just being introduced. Later, Copeland (1946) suggested that the problem of recidivism in the Army was closely related to the problem of the dullard. This somewhat overstates the case. Though the incidence of dullness is higher in recidivist groups than in general delinquent or non-delinquent groups (Table I), only a minority of all dull soldiers become delinquent.

The distributions of all three samples in Table I are initially slightly skewed in a negative direction since none include officers or N.C.O.s. The non-delinquent sample is matched with the recidivist group for corps as well as for rank and is thus more skewed than would be expected. Notwithstanding the

TABLE I

Military Groups Classified by General (Non-verbal) Intelligence Grade

Military Groups	Intelligence Grades (Percentages)					
	1 Superior	2 Above Average	3+ Average	3- Average	4 Below Average	5 Dull
Theoretical army distribution	10	20	20	20	20	10
Non-delinquents (N=150) ..	3	14	19	23	26	15
Delinquents (N=500+) ..	6	14	20	22	24	14
Recidivists (N=147) ..	0	6	8	28	35	23

peculiarity of the non-delinquent sample, the Table shows clearly enough that dullness is not necessarily associated with delinquency or recidivism.

Hubert (1944) examined 105 soldiers who had been sentenced to detention for desertion. Of these, 76 had "no neurotic tendency", 20 "a slight neurotic tendency", and 9 "a marked neurotic tendency". The intelligence distribution of this group approximated that of the Army as a whole. He concluded that the problem of desertion was essentially military rather than medical in nature.

Penton (1944) reported on 103 soldiers under sentence, most of whom were deserters. He classified 22 as having "good normal" personalities, 10 had "normal" personalities but had shown previous neurotic reactions, 37 had "adequate" personalities with a history of continuing neuroticism, 13 were mental defectives, 16 were classified "immature", and 5 were diagnosed "psychopathic, with anti-social trends". Despite this classification, all but three of the men were regarded as psychiatrically fit for some form of full or limited military service.

In 1946, Penton studied some 2,000 deserters and stated that 43 per cent. had normal basic personalities. The remaining 57 per cent. were diagnosed as: immature—21 per cent., dullards—5 per cent., psychopaths—5 per cent., inadequate personalities—24 per cent. Penton's conclusion that personality instability plays an important part in the aetiology of desertion suggests that, contrary to Hubert's view, desertion is at least as much a psychiatric problem as a military one. As desertion and absence without leave are the most common of the serious military offences, this finding is of particular interest.

Special Training Units were established during wartime to deal with young soldiers who had failed to adjust satisfactorily to Army life. Owing to careful selection for these units few of the offenders posted to them were found to suffer from gross mental disability, but Trenaman (1952) points out that very few of the men were "in any way normal people, with a balanced attitude to life".

Despite the difficulty of precise clinical differentiation in this field, the investigators cited here have had to make some form of classification for descriptive, administrative, and corrective purposes. The most useful of the classificatory schemes was devised by Penton (1944, 1946) who used it as a basis for his recommendations concerning the military delinquents he examined. The main clinical characteristics that have been determined in military delinquent groups may conveniently be summarized under his headings here condensed into five categories:

(1) *Normal, adequate personality.* Men with adequate basic personalities may react with delinquent behaviour to specific stress situations arising, for example, from domestic difficulties or from faulty allocation to employment (Davis, Wolman, Berman and Wright, 1945). The motivation for the delinquent

behaviour of basically normal soldiers is usually conscious and deliberate and may often be due as much to uncomplicated irresponsibility or self-seeking as to personal anxiety (Davidoff, 1947).

(2) *Inadequate and neurotic personality.* When faced with the rigid institutional demands of the Army, men with continuing histories of social failure and non-adaptive reactions to stress may present psychosomatic symptoms in conjunction with their delinquency. Their outlook may be paranoid, and their malingering, self-inflicted wounds, and suicidal attempts may be seen as futile attempts to revenge themselves on society as well as conscious efforts to avoid military duty (Weinberg, 1945). That rather rare group of delinquents whose behaviour is characterized by classical psychoneurosis may be considered here as a sub-category. Their deviance represents a symptomatic and compulsively repeated response to their unconscious conflicts, whereas the inadequate tends to compensate through delinquency for his inferiority or deprivation (Rees, 1949).

(3) *Mental dullness.* The dullard lacks the capacity for making new adjustments readily, and if the anxiety aroused by the conflict between his rigid habits and the military requirements is too great, he may seek escape through A.W.O.L. or desertion (Heine, 1945). Dull soldiers, being eager to gain acceptance instead of ridicule, can be led into trouble rather easily. The most obvious explanation of many offences committed by dullards is that such men have failed to understand either the regulations or the reasons for them (Rees, 1945).

(4) *Immaturity.* A number of soldiers who commit military offences may be classified as immature. They are adolescents without strong identifications, and their reactions are basically childish. Trenaman (1952) indicates that much of their external "tough" behaviour is a façade concealing feelings of insecurity and inferiority. The insecurity of these psychologically under-developed youths may often be related to early emotional deprivation and a consequent failure to introject mature standards of authority and discipline.

(5) *Psychopathic personality with anti-social trends.* There seems now to be sufficient agreement concerning the aetiology, psychopathology, and symptomatology of psychopathy to enable reasonably clear identification of this abnormality (Darling, 1945). The psychopath's continuing endeavour to gain immediate and impulsive gratification despite repeated punishment is characteristic of him (Szurek, 1942). In units, they not only get into trouble themselves but tend also to influence others of less intelligence, or those who may be unsettled (Lewis, 1951). Psychopaths with paranoid or aggressive trends might do well in action, but Pozner (1950) points out that as only a small percentage of soldiers became engaged in hand to hand combat, and then for relatively short periods, psychopaths are probably an over-all liability. The liability would be particularly great in peace, and these men would be unreliable at all times. Psychopaths of this kind are prone to commit offences of violence and to be wilfully disobedient and destructive (Dunn, 1941; Cowan, 1945).

In civil life many unstable characters function well, and only the failures are seen in prisons and hospitals (Kardiner and Spiegel, 1947). This is probably just as true of the military population though soldiers form a selected group. Even our information about the incidence of mental disability among military delinquents is limited for only those referred by commanding officers or unit medical officers receive a psychiatric examination. In psychiatric practice it is axiomatic that almost anyone will break down in situations of sufficiently

severe or prolonged stress (Strecker and Appel, 1945), though the unstable and seriously predisposed will break soonest. Similar considerations probably apply in the field of military delinquency where the most seriously predisposed may be those suffering from some psychiatric disability.

Many recruits who may be predisposed to delinquency are diverted at an early stage. If recruits display gross abnormality they are usually excluded by routine pre-enlistment medical examination. Further screening of all others takes place at depots and training units. There recruits are given intelligence and aptitude tests and interviewed by personnel selection officers who make employment recommendations for each man. At this stage soldiers may be referred for psychiatric examination. Typical cases for referral are men with very low test scores, stammerers, illiterates, men who have attended special schools, and those with a history of psychiatric illness or with apparent symptoms of abnormal behaviour (Privy Council Office, 1947). Those unfit for any form of military employment may then be hospitalized or discharged, and mild psychoneurotics and dull but stable soldiers can be given special attention in allocation to suitable employment. There is no doubt that this careful allocation has prevented many men from becoming psychiatric or disciplinary casualties.

A number of conclusions can be drawn with reasonable confidence from the foregoing material. Although the Army's screening processes exclude most recruits with severe psychiatric disabilities, some escape detection. Some others, whose personality disorders might not be recognized in the more flexible civilian life, may become incapacitated under the increased stress of military conditions. Mental abnormality—psychoneurosis, psychopathy, and dullness—is frequently closely associated with military delinquency, though such abnormality does not necessarily lead to delinquent behaviour. The evidence suggests that the incidence of psychiatric disability is higher among soldiers under sentence than among the military parent population. On the other hand, it is clear that by no means all delinquent soldiers suffer from some form of psychiatric disability.

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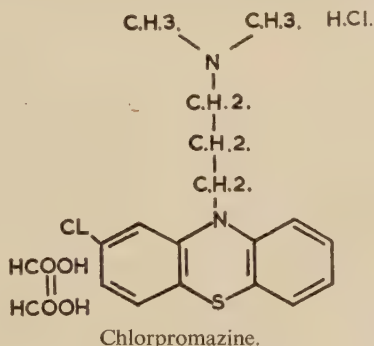
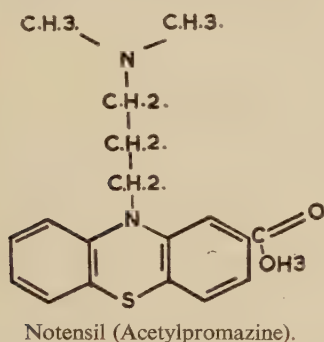
THE EFFECTS OF NOTENSIL IN CHRONIC MENTAL ILLNESS

By

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ANOTHER "tranquillizing" drug known as "Notensil" has recently been introduced to this country. It is a phenothiazine derivative, acetylpromazine, closely related chemically to chlorpromazine but without the chlorine atom which has been thought to cause the undesirable side effects of the promazine group. The structural formula of "Notensil" compares with that of chlorpromazine as follows:



The drug was first introduced in France where workers claim:

1. "Notensil" and chlorpromazine have similar, if not identical, therapeutic indications. "Notensil" is effective in relieving hostility, apprehension and anxiety in the major psychoses.

2. "Notensil" is more active—an equivalent therapeutic dose being 50–75 per cent. of chlorpromazine.

3. It is less toxic than any other drug in the category of tranquillizers. The minor side effects including tachycardia, gastric upset, etc., have not been observed with "Notensil".

The purpose of this trial was to investigate these claims.

MATERIAL AND METHOD

Fifty patients (29 female and 21 male) were given "Notensil" orally. They were all suffering from long-standing mental illnesses and their present stay in hospital ranged from 1 to 36 years with an average of 12 years. The average age was 43 years. Forty-seven suffered from a schizophrenic type of illness, two of these being also mentally defective, one was a schizoid psychopath, one an epileptic idiot and one a chronic hypomanic. The majority had previously had some form of physical treatment and all had previously received other tranquillizing drugs, many with benefit. Practically all the patients had taken part in two former controlled trials with other drugs.

The tablets were given three times daily. Treatment was commenced with 30 mg. daily which was increased to 75 mg. daily after four days and to 150 mg. daily after a further five days. This dose was maintained for four weeks, but in the patients who showed aggressive trends (34), it was increased to 225 mg. daily after two weeks. Finally a reduced dose of 75 mg. daily for 12 days. "Notensil" was thus given for seven weeks in all. It had been intended to follow this by the substitution of a placebo for the drug, but, in view of the disappointing results, it was not considered necessary to proceed with this.

RESULTS

The individual results were assessed by myself with the assistance of the sisters or charge nurses who were in daily contact with the patients. The nursing staff have now had considerable experience in this hospital in the investigation of the effects of various new drugs.

Table I summarizes the results of treatment. Only 2 patients (4 per cent.) benefited from the drug whereas 22 patients (44 per cent.) were definitely and significantly worse whilst "Notensil" was being administered.

TABLE I
General Results from Treatment with Notensil

	Female	Male	Total
Much improved	0	0	0
Improved	1	1	2 (4%)
No change	14	12	26 (52%)
Worse	12	8	20 (40%)
Much worse	2	0	2 (4%)
Total	29	21	50

Table II shows a more detailed analysis of the effects of "Notensil" on various signs and symptoms. The most notable feature is the increase in overactive, aggressive and impulsive tendencies produced by the drug. Thirty-four patients were classed as "aggressive" when treatment was commenced and in 18 (53 per cent.), the aggression became worse, 16 were "overactive", the overactivity increasing in 11 (69 per cent.), and 29 were "impulsive", 11 (38 per cent.) becoming worse.

TABLE II
Analysis of the Effects of Notensil on Certain Signs and Symptoms

Symptoms and Signs	Present at Onset	Improved	No Change	Worse
Aggression	34	1 (3%)	15 (44%)	18 (53%)
Delusions	37	0	28 (76%)	9 (24%)
Hallucinations	36	1 (3%)	24 (67%)	11 (30%)
Severe withdrawal	16	2 (13%)	13 (81%)	1 (6%)
Retardation	17	2 (12%)	14 (82%)	1 (6%)
Overactivity	16	0	5 (31%)	11 (69%)
Depression	8	0	8 (100%)	0
Elation	6	0	4 (67%)	2 (33%)
Confusion	32	0	28 (88%)	4 (12%)
Impulsiveness	29	1 (3%)	17 (59%)	11 (38%)
Faulty habits	7	0	7 (100%)	0

A heightened restlessness and agitation appeared in many. In addition, one patient who had not previously been aggressive and impulsive became so, one patient became overactive for the first time and one patient developed faulty habits. On the other hand, there was no significant improvement in patients who were withdrawn and retarded.

The majority of the patients who deteriorated under the drug, had returned to their former states within two weeks of stopping it.

There was an average drop of 7 mm. in the systolic blood pressure during the administration of the drug. There was no significant alteration in the average diastolic blood pressure, the pulse rate or in weight during the course of treatment.

TOXIC AND SIDE EFFECTS

No toxic or side effects were encountered apart from one patient who had a syncopal attack after being on the drug for 34 days. Treatment was suspended for four days and then resumed with no further ill effects. This incident occurred in a male patient aged 50 years, who was the only one to show a large drop in blood pressure, his B.P. falling from 180/85 to 110/70 in the seven weeks of treatment.

DISCUSSION

"Notensil" was found to be of no value in the treatment of this series of chronic psychotic cases. On the contrary, the majority of patients with aggressive and overactive tendencies became worse. In view of this increase in overactivity, it might have been expected that the drug would benefit retarded and withdrawn patients but this was not found to be the case.

It is claimed that "Notensil" has a depressant action on the central nervous system, centred on the reticular substance of the midbrain and that this allows the cortex to function normally though protected from hyperactivity of the midbrain. This is obviously not always the case. It would appear that, where there is a tendency to aggression or overactivity, "Notensil" further diminishes any cortical inhibitory action.

The marked absence of any toxic or side effects is possibly a hopeful pointer to the finding of a phenothiazine drug which will combine this property of "Notensil" with the more active tranquillizing effects of other members of the group.

SUMMARY AND CONCLUSIONS

1. The effects of a new drug "Notensil" (acetylpromazine) on 50 chronic psychotic patients are described.
2. Only 4 per cent. of the patients benefited from the drug, whereas 44 per cent. were definitely worse.
3. "Notensil" is considered to be of no value in the treatment of chronic psychosis. Moreover, it would appear to be distinctly harmful where there is any tendency to aggressive, impulsive or overactive behaviour.

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EFFECTS OF ELECTRICAL STIMULATION OF THE BRAIN ON THE CONCENTRATION OF ADRENALINE-LIKE SUBSTANCES IN HUMAN PLASMA

By

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CEREBRAL electrostimulation of subconvulsive intensity causes an increase in the concentration of adrenaline-like substances in the blood (Weil-Malherbe and Bone, 1952b). This increase becomes more pronounced as the pulse repetition rate is raised from 200 pulses per second (p.p.s.) to 500 p.p.s., in contrast to the motor, sensory, and respiratory effects which diminish under these conditions (Montagu, 1955). The increasing response to the higher repetition rates showed no sign of reaching a peak in these experiments, except in one case, and since it was not possible with the existing apparatus to exceed 500 p.p.s., the optimal conditions for eliciting the response remained undetermined. With the aid of a more versatile stimulator the adrenaline-frequency relationship has subsequently been studied more extensively, and the results are presented in this article.*

APPARATUS

The stimulating current consisted of unidirectional, rectangular impulses, which were generated by means of a multivibrator. Instead of the usual arrangement for selecting the pulse duration, provision was made for controlling the pulse/interval ratio. This was independent of the repetition rate so that any change in the latter was accompanied by an inversely proportional change in both pulse duration and interval. Consequently, at any given peak current the average output remained the same regardless of the repetition rate. The conditions were therefore similar to those which obtained in the previous study (Montagu, 1955).

The *pulse repetition rate* was variable from 10 to 8,000 p.p.s. Ten steps covered the basic range from 10 to 80 p.p.s., and these rates could be multiplied either ten-fold or one hundred-fold by means of another switch. The tolerance of the repetition rate was about 5 per cent.

The *pulse/interval ratio* was variable in eleven steps from 1:4 to 1:99, i.e. the pulse duration was equal to 1/5th of the total cycle at the lowest ratio and to 1/100th at the highest extreme. At any given setting the ratio remained constant to within about 10 per cent. regardless of the pulse repetition rate.

The *intensity* was continuously variable from zero up to 600 mA. peak. It remained independent of the load provided that the output voltage did not exceed 150 volts. Both peak and average currents were metered separately.

* A grant in aid of equipment was received from the Medical Research Council and is gratefully acknowledged. The stimulator was developed by the Multitone Electric Company Limited, London, to the author's specification.

Only a few of the available conditions were used in the experiments. Four pulse/interval ratios were selected for investigation, namely, 1:9, 1:24, 1:49 and 1:99, and the frequency spectrum was studied from 400 p.p.s. to 2,000 p.p.s. The pulse durations under the various combinations of these conditions are given in Table I.

TABLE I
Pulse Duration (μ sec.) at the Various Repetition Rates and Pulse/Interval Ratios

Repetition Rate (p.p.s.)	Pulse/Interval Ratio			
	1 : 9	1 : 24	1 : 49	1 : 99
400	250	100	50	25
500	200	80	40	20
600	167	67	33	17
800	125	50	25	13
1,000	100	40	20	10
1,200	83	33	17	8
1,500	67	27	13	7
2,000	50	20	10	5

MATERIAL AND METHODS

The subjects were sixteen men who were suffering from anxiety states. Their ages ranged from 20 to 43 years.

The samples of blood were collected by withdrawing 15 ml. into 5 ml. of a mixture of ethylenediamine tetra-acetate and thiosulphate (Renton and Weil-Malherbe, 1956). After an initial sample had been taken, the subject was given 0.75 gm. of thiopentone by slow intravenous injection. A second blood sample was taken about a minute later. The scalp was then cleaned just above each ear, electrode jelly was rubbed in, and the electrodes were applied. These were circular metal discs 3 cm. in diameter, which were covered with chamois leather soaked in saline. The required pulse/interval ratio was selected, and the current was slowly raised. In half of the experiments stimulation was begun with a repetition rate of 400 or 500 p.p.s. and this was increased step by step at intervals of $1\frac{1}{2}$ -2 minutes; in the other half the repetition rates were applied in descending order. In the latter cases, the subject's tolerance to stimulation at 400 p.p.s. was determined in a rapid preliminary trial, since previous studies had shown that the motor, sensory, and respiratory effects of the current were more marked at the lower frequencies (Montagu, 1955). In each case the intensity was raised to the highest point compatible with free respiration, and it was then maintained at this level throughout the experiment. The individual tolerance was found to be remarkably constant from subject to subject, but it varied considerably for different pulse/interval ratios. Thus, with ratios of 1:9, 1:24, 1:49 and 1:99, the respective peak currents were, with one exception, 40-50 mA., 80 mA., 100 mA., and 140-150 mA. In the exceptional case (Case 12), the peak current was raised to 250 mA. with a pulse/interval ratio of 1:99 without producing any observable change in the respirations.

A sample of blood was taken during stimulation at each pulse repetition rate. In every instance at least $1\frac{1}{2}$ minutes was allowed to elapse after a change of repetition rate before the withdrawal of the next sample was begun. Previous

experiments had indicated that this period was sufficient to allow the blood adrenaline concentration to reach the new level (Montagu, 1955).

The blood samples were centrifuged, and the plasma adrenaline concentrations were estimated at Runwell Hospital by the method of Weil-Malherbe and Bone (1952a, 1953).

RESULTS

The results of the experiments are given in Table II, which shows that in

TABLE II
The Effects of Electrostimulation on the Blood Adrenaline Concentration

Case	Pulse Rate Sequence*	Pulse/Interval Ratio	Adrenaline Concentration $\mu\text{g/l}$		Increase in Adrenaline Concentration above "Basal" Level During Stimulation $\mu\text{g/l}$							
			Initial	"Basal" (After Thiopentone)	400 p.p.s.	500 p.p.s.	600 p.p.s.	800 p.p.s.	1,000 p.p.s.	1,200 p.p.s.	1,500 p.p.s.	2,000 p.p.s.
1	A	1:9	1.52	0.77	0.13	0.73	0.24	0.32	0.59	0.28	0.40	
2	A	1:24	1.03	0.35	0.16	0.62	0.97	0.66	0.75			
3	A	1:49	0.89	0.35	0.66	1.17	0.78	0.63	0.64	0.49		
4	A	1:99	1.20	0.62	0.94	0.95	0.62	0.86	0.62	0.63	0.75	0.67
5	D	1:9	1.08	0.52	0.95	1.58	0.42	0.43	0.18	0.15	0.09	
6a	D	1:24	0.87	0.26	0.57	0.61	0.52	0.30	1.14	0.31	0.15	
6b	A	1:24	1.09	0.15	0.32	0.74	0.44	0.76	0.72	0.65	0.57	0.60
7	D	1:49	0.88	0.44	0.23	0.36	0.31	0.09	0.09	0.04	0.01	
8	D	1:99	1.00	0.19	0.59	0.76	0.36	0.38	0.20	0.25	0.07	
9	A	1:9		0.43		0.78	0.56	0.37	0.50			
10	A	1:24		0.32		0.97	0.86	0.68	0.63			
11	A	1:49		0.35		0.56	0.26	0.47	0.31			
12	A	1:99		0.50		0.47	0.66	0.44	0.11			
13	D	1:9		0.32		0.29	0.18	0.11	0.14			
14	D	1:24		0.50		0.33	0.10	0.05	0.05			
15	D	1:49		0.56		0.27	0.01	0.07	0.33			
16	D	1:99		0.72		0.56	0.30	0.26	0.10			
Mean			1.06	0.43	0.51	0.69	0.44	0.40	0.42	0.35	0.29	
Standard error			± 0.07	± 0.04	± 0.10	± 0.08	± 0.06	± 0.06	± 0.08	± 0.08	± 0.11	

* A=Ascending order; D=Descending order.

the first nine cases the injection of thiopentone caused a brisk fall in the circulating adrenaline. This effect has been found to occur with such constant regularity (Weil-Malherbe and Bone, 1952a; Weil-Malherbe, 1955; Montagu, 1955) that the collection of the initial blood sample was discontinued in the later experiments. The first specimen was taken soon after the induction of anaesthesia, and the effect of each period of stimulation is given in the Table in terms of the increase in adrenaline above this "basal" level.

1. Effect of Pulse Repetition Rate

In twelve of the seventeen experiments the blood adrenaline concentration reached its highest point during stimulation at 500 p.p.s., and in two more cases the levels at this rate were only just below the highest values attained, which occurred in response to 800 p.p.s. (Case 6b) and 1,000 p.p.s. (Case 15). In the remaining three experiments indisputable peaks were encountered twice at 600 p.p.s. (Cases 2 and 12) and once at 1,000 p.p.s. (Case 6a).

A moderate amount of haemolysis in this last sample cast some doubt upon the significance of the high concentration which was found in it, and it prompted a further experiment in the same subject. On the second occasion stimulation had similar effects at 500, 800 and 1,000 p.p.s. Nevertheless, the amount of haemolysis which occurred in the first experiment was not considered

sufficient to account for the whole of the observed increase at 1,000 p.p.s., and none was seen in the corresponding sample in Case 15, which also yielded a high value.

The mean figures for all the experiments show a maximum response at 500 p.p.s. with decreasing effects at higher and lower repetition rates. To test the significance of this peak the effects of stimulation at 500 p.p.s. were compared with those of the two adjacent pulse rates. In examining the difference between 500 and 400 p.p.s., only the first nine experiments were considered in order to take advantage of the pairing of the observations in the same subjects. The mean difference in response at these two rates was found to be $0.33 \mu\text{g/l}$ of adrenaline, which was significant at the 1 per cent. level of confidence ($t=4.074$). Between 500 and 600 p.p.s. the mean difference in effect in all seventeen cases was $0.25 \mu\text{g/l}$, and this also was significant at the 1 per cent. level ($t=3.309$).

2. *Effect of Pulse Rate Sequence*

In Cases 1-4 and 9-12 the pulse repetition rates were taken in ascending series; in the remaining experiments the reverse order was adopted, with the exception of the re-test in Case 6. In this way the effects of the waning anaesthesia as each experiment progressed were minimized in the mean results.

To study the influence of this effect on the results, the difference between the responses to stimulation at 500 and 1,000 p.p.s. was calculated in each case. It was found that in the "ascending" group there occurred a mean decrease in blood adrenaline of $0.24 \mu\text{g/l}$ as the repetition rate was raised from 500 to 1,000 p.p.s. through the intermediate stages. In the "descending" series, on the other hand, the adrenaline concentration rose by an average of $0.32 \mu\text{g/l}$ as the pulse rate was reduced from 1,000 to 500 p.p.s. When the t -test was applied to these figures, the difference between them was found to be insignificant ($t=0.407$; $P>0.05$), which suggests that the effects of the changing depth of anaesthesia were small in comparison with the effects of stimulation.

Further confirmation was obtained by comparing the two groups with respect to their responses to the optimal repetition rate, 500 p.p.s. In the "ascending" series this caused a mean increase in the blood adrenaline of $0.78 \mu\text{g/l}$; the corresponding figure for the "descending" series was $0.60 \mu\text{g/l}$. The difference was tested and was found to be not significant ($t=1.096$; $P>0.05$).

3. *Effect of Pulse/Interval Ratio*

An increase in the pulse/interval ratio involves a decrease in the pulse duration at any given repetition rate. Furthermore, in order to keep the average current constant throughout each experiment, an increase in the repetition rate was also accompanied by a proportional abbreviation of the pulses. Consequently, as shown in Table I, the pulse duration was minimal at the higher repetition rates and with a pulse/interval ratio of 1:99. It seemed reasonable to suppose that such pulses, with a duration of only a few microseconds, might cease to act as effective stimuli, hence causing a shift of the frequency-adrenaline relationship in the direction of the lower repetition rates.

Table II, however, shows no obvious difference between groups of experiments in which the pulse/interval ratio varied widely. There were only five occasions on which the effects of stimulation at 500 p.p.s. were equalled or exceeded at higher pulse rates. This occurred three times with a ratio of 1:24 and once each with ratios of 1:49 and 1:99. Conversely with expectation, no

increase in effect at the higher rates was observed with the lowest ratio when the pulses were longer.

DISCUSSION

Cerebral stimulation causes a progressive increase in the concentration of adrenaline-like substances in the plasma as the pulse repetition rate is raised to 500 pulses per second, the peak and average currents being kept constant (Montagu, 1955). In the present study this range has been considerably extended under otherwise similar conditions, and a diminution in the effects has been observed. The greatest liberation of adrenaline occurred in response to 500 pulses per second.

This conclusion is in good agreement with the observations of Mihailovic and Delgado (1956) in monkeys. Using three peripheral reactions as signs of autonomic response, they found that the threshold was lowest with repetition rates of about 250–500 p.p.s. These rates were higher than those which had been previously reported.

An earlier report (Montagu, 1955) has emphasized that a repetition rate of 500 p.p.s. was considerably higher than that which aroused the greatest motor and sensory responses. An increase in the repetition rate from 200 to 500 p.p.s. resulted in a progressive decrease in the ability of the current to cause tonic contractions, respiratory embarrassment, and pain. The adrenaline level was therefore rising as the outward signs of stimulation diminished.

It has recently been pointed out (Anonymous, 1956) that there are certain obstacles to the acceptance of the results obtained by Weil-Malherbe and Bone (1952a) and Weil-Malherbe (1955) with their method of estimating the circulating adrenaline. The uncertainty has arisen as a result of discrepancies between the biological and chemical methods of assay, the relative merits of which will not be considered here. Weil-Malherbe's most recent work (1956, 1957) supports the view that he is, in fact, measuring adrenaline itself. Even if it subsequently develops that this is not the case, it is probable that the substance or substances concerned are closely associated both with adrenaline and with sympathetic activity.

SUMMARY

Electrical stimulation of the brain through surface electrodes causes an increase in the concentration of adrenaline-like substances in human plasma. This effect becomes more pronounced as the pulse repetition rate is raised up to 500 pulses per second (Montagu, 1955).

It has now been established that the greatest increase in the adrenaline level occurs in response to 500 pulses per second; above this point the effect declines. The significance of this peak has been demonstrated.

The frequency response of the adrenaline level differs considerably from the motor and sensory responses to stimulation. These increase with each decrease in repetition rate below 500 pulses per second.

ACKNOWLEDGMENTS

I wish to express my thanks to Dr. H. Weil-Malherbe for the biochemical estimations upon which this work so largely depended. Also to Dr. L. S. Davies, Deputy Director of Roffey Park Rehabilitation Centre, for again providing the clinical material and facilities. I am indebted to the Physician Superintendent, Dr. R. Ström-Olsen, for permission to publish this report.

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INVESTIGATION OF AMINO ACIDS IN A PERIODIC CATATONIC PATIENT

By

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THE nitrogen retention, and especially the periodically changing excretion of urea, seems to be a characteristic symptom in periodic catatonia (1). We therefore made it our aim to investigate the amino acids in the urine, and later also in the blood and cerebro-spinal fluid (C.S.F.), throughout the lucid interval and the catatonic phase.

We also arranged to treat the patient with A.C.T.H. and adrenaline, and finally to compensate the condition with thyroxine while at the same time tracing the amino acids, hoping to get a clue regarding which metabolic processes or chains may be deficient in periodic catatonia, especially with regard to the synthesis of urea and the detoxication of ammonia.

EXPERIMENTAL

A. Methods: For 2½ years we have daily investigated the patient clinically (as described by R. Gjessing: pulse, temperature, weight, sleep, psychiatric evaluation and description of behaviour). For periods of weeks and months we have examined 25 amino acids semi-quantitatively by paper chromatography (2).

Serum and C.S.F. were ultra-filtered by a method based on those of Greenberg and Gunther (3) and Dent and Schilling (4). The collodion membrane was made with 6 per cent. polynitrocellulose in ethanol-ether (1:1 v/v) to which 4 per cent. (v/v) ethyleneglycol was added.

Samples (1 ml.) of native C.S.F., ultrafiltered C.S.F. and ultrafiltered serum, as well as urine samples, were desalted electrolytically, with Dent's apparatus and analysed both before and after hydrolysis. The urine was diluted to contain 0.5 mg. N/ml. Hydrolysis was carried out with 6 N-HCl in a closed glass tube in boiling water for one hour.

The amino acids were separated in a thermostatically regulated room by two dimensional paper chromatography. The solvents were phenol: water 4:1 (v/v), and 2,4,6-collidine: 2,6-lutidine: distilled water 2:1:2 (v/v) saturated with water at 20° C., on Whatman paper No. 4 (45 × 50 cm.). The paper was treated with ninhydrin and dried by warm air (40° C.) for 30 minutes and the amino acids were identified by their characteristic colours when dry. After 24 hours the intensity of the colours was estimated by eye on a 0-10 scale when viewed at constant illumination by the same person each time. The area of the spots was measured by planimeter. The product of intensity and area was used as an index of the amino acid content.

By testing different equimolar solutions of amino acids it was possible to show that one group of amino acids (glutamine, glutamic acid, serine, taurine, citrulline, valine and alanine) gave a bluish purple colour which followed Beer's law. Another group (aspartic acid, *beta*-alanine, leucine, glycine, threonine, lysine, arginine, tyrosine and ornithine), giving colours deviating from bluish purple, did not follow Beer's law when in small concentrations (below 10 µg.), but showed good correspondence between added and estimated amounts in larger concentrations above 10 µg. It was therefore possible to express our readings in µg., but as we were only interested in the relative changes during the illness, and during treatment and recovery, we have, in our curves, kept to our primary observations.

Pond (5) has estimated the error by this semiquantitative method to be ±30 per cent. We have used a similar method on only one patient, on constant fluid diet, from day to day or from week to week, and we always carried out hydrolysis as a control: furthermore the analyses were all done by only one of us (A. Bernhardsen). Therefore we believe that the error may have been reduced to ±10-15 per cent. in our work.

During the periods when the amino acids were investigated the patient was living on a standard fluid diet (H diet): 2 egg yolks, 1,100 ml. milk, 365 ml. cream (10 per cent. fat), 150 g. sugar, 2 g. NaCl and in addition mineral salts (Fe, Cu and Mn) and vitamins.

CASE REPORT

The patient was born 25 March, 1927. A brother and an aunt of the patient have been mentally ill and the brother has been leucotomized. The mother is a typical syntonous pyknic and the father is a short leptosome stable type.

Premorbid: the patient was extrovert, jovial, clever in working, socially well adapted, childish, easy going and intellectually normal. It has not been possible to find any exogenous causes for her mental disease.

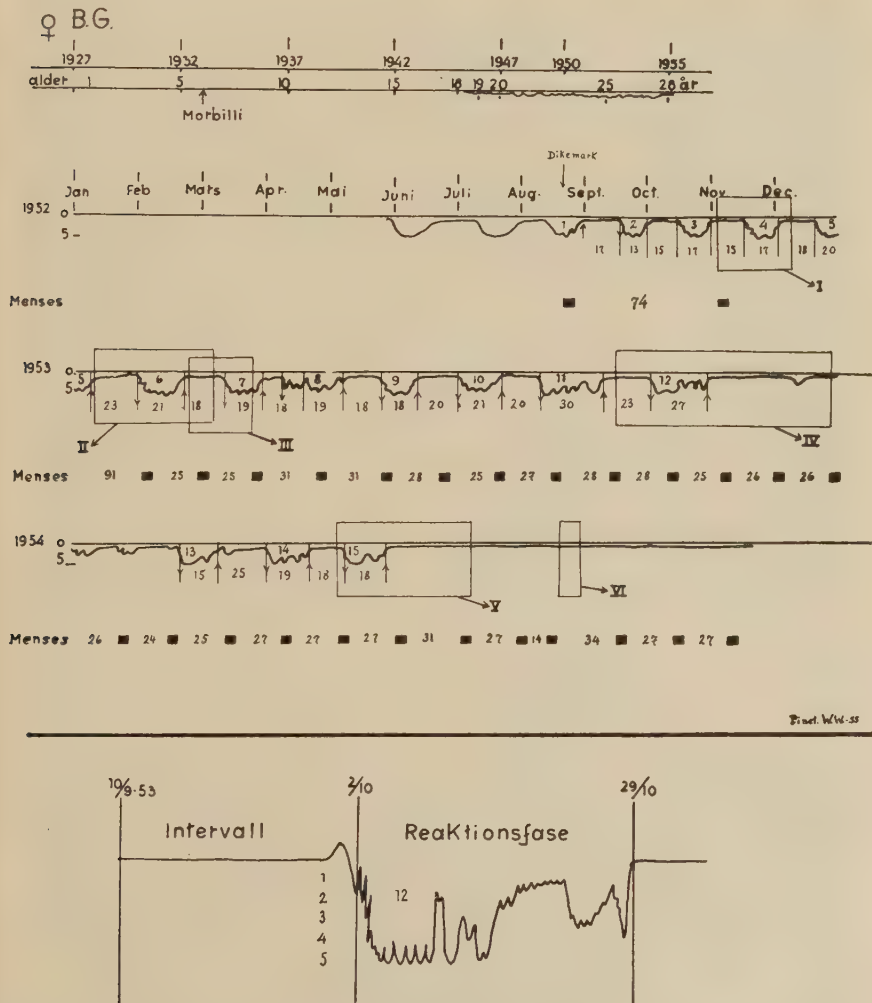


FIG. 1.—Upper part: Picture of the catatonic phases and intervals a few months before and during the periods of investigation, correlated with menses: Periods I and II amino acids in urine, IV, V and VI amino acids in serum and C.S.F., before (IV), during and after treatment with thyroxine (V) and during A.C.T.H. test (VI). Lower part: an illustration of the patient's power of concentration during interval and catatonic phase.

The patient has been mentally ill since 1946 and hospitalized about one year and treated with 50 E.C.T. before she arrived at Dikemark Hospital in August, 1952. She was a typical pyknic and cyclic. The physical and neurological investigation did not show any abnormalities. The pneumoencephalogram was normal.

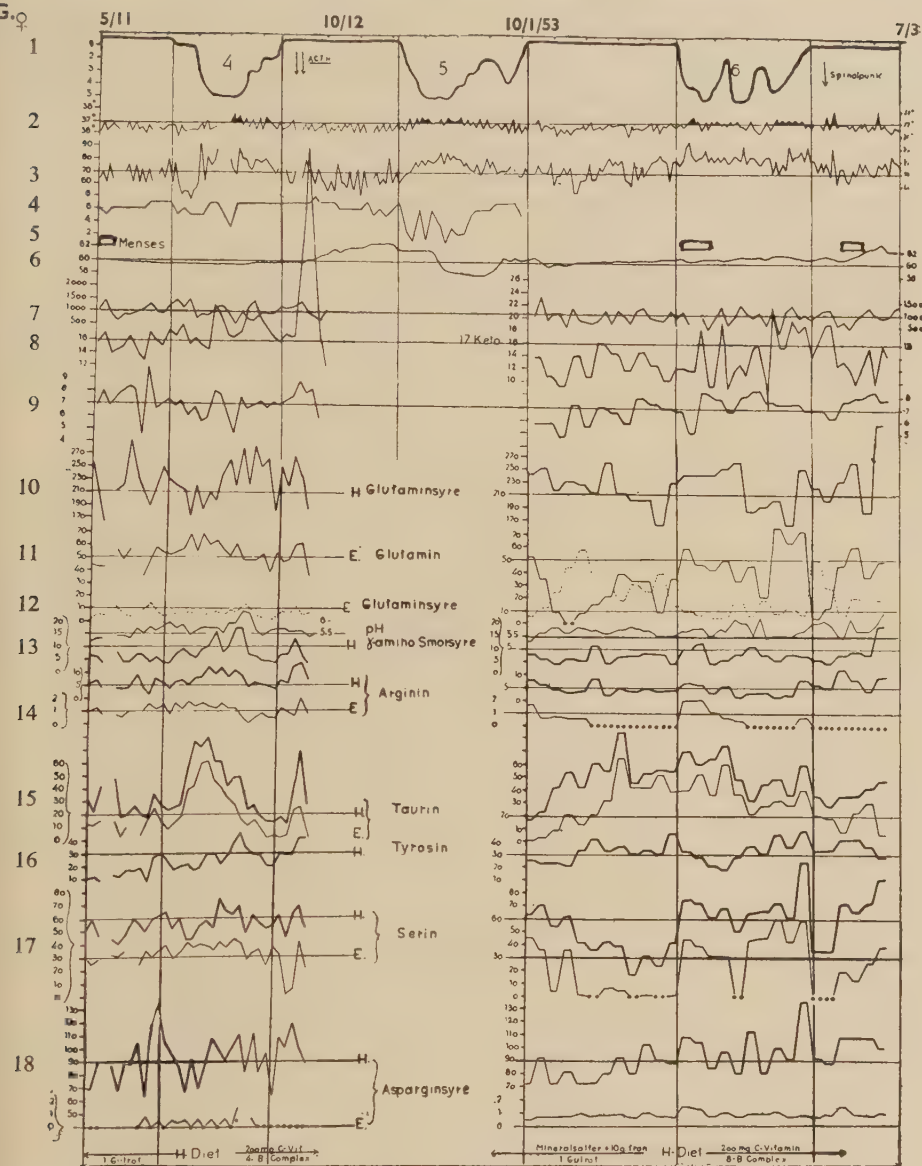
The pattern of the patient's illness was 15 catatonic stupor phases lasting from 13 to 30 days, but most often lasting 18 days. These phases were separated by intervals (17-23 days) in which the patient showed few pathological symptoms (see Fig. 1).

The *catatonic phases* each time showed the same sequence of symptoms, though with certain deviations which may have been caused by the interference



FIG. 2.—The first two pictures present the patient during the lucid interval. The next four pictures (from 24.5.1953 to 1.6.1953) illustrate the patient as she gradually goes into stupor. The last picture is taken ten days later when the patient had recovered from the psychotic attack.

B.G.♀



- | | | |
|------------------------|---------------------|-----------------------|
| 1. Concentration power | 7. Diuresis | 13. Aminobutyric acid |
| 2. Temperature | 8. 17-ketosteroids | 14. Arginine H+E |
| 3. Pulse | 9. Total N in urine | 15. Taurine H+E |
| 4. Sleep | 10. Glutamic acid H | 16. Tyrosine H |
| 5. Menses | 11. Glutamine | 17. Serine H+E |
| 6. Weight | 12. Glutamic acid E | 18. Aspartic acid H+E |

FIG. 3.—These curves illustrate three catatonic phases (4, 5 and 6) correlated with temperature, pulse, sleep, weight, diuresis and the urinary excretion of 17-ketosteroids, total Nitrogen as well as 8 amino acids.

The first period of investigation from 5.11.1952 to 10.12.1952 shows a very different pattern in all the curves compared with the second period of investigation from 10.1.1953 to 7.3.1953. This difference may be due partly to absence and presence of menses, and partly to the increased vitamin B complex in the diet during the second period of investigation.

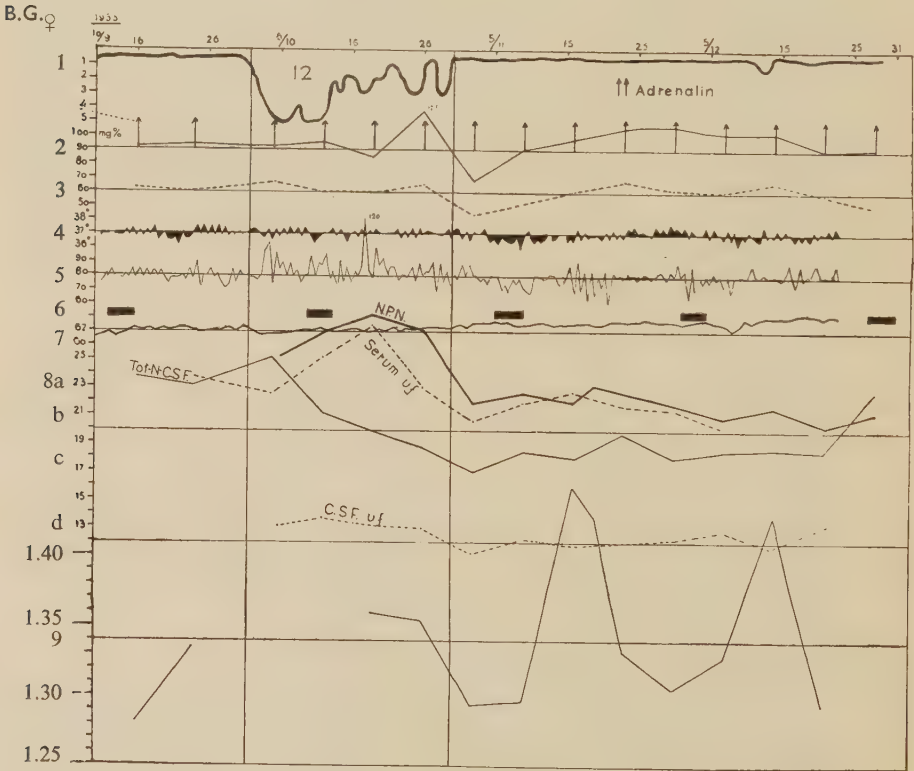
In the interval after the catatonic phase No. 4, the patient was given A.C.T.H. i.m. (150 mg.). The 17-ketosteroids increased from 16 to 46 mg. in the urine. Taurine had also a sharp peak.

of menses: (1) The patient became emotionally unstable, thereafter (2) cranky, dysphoric and evasive and gradually (3) silly with poor powers of concentration and finally (4) amimic, mute, absent minded, apathetic with spells of temper tantrums. In addition soiling with urine and excrement. After 8-10 days the symptoms gradually disappeared (see Figs. 1 and 2).

Physically: nearly normal temperature, rapid pulse, interrupted sleep, unwillingness to eat, constipation and retention of urine, and loss in weight when she was not on the standard diet.

In the lucid *interval*, however, the patient was clear, fully orientated and occupied with sewing and reading and slightly hypomanic.

Physically: Subnormal temperature, slow pulse, decreased B.M.R., sleeping well, good appetite, no urine retention or constipation and slight increase in weight.



1. Power of concentration
2. Blood sugar
3. Spinal sugar
4. Temperature
5. Pulse
6. Menses

7. Weight
8. (a) NPN
- (b) Total N u.f. serum
- (c) Total N C.S.F.
- (d) Total N u.f. C.S.F.
9. Total N serum in gm. %

mg. %

FIG. 4.—These curves illustrate when the patient had her lumbar punctures in relation to the catatonic phase and adrenaline tests.

Blood and spinal fluid sugar did not change very much. The temperature curve is not increased during the catatonic phase. The menstruation is pretty regular. The weight is slowly increasing. NPN and u.f. serum N reached their maximum in the second half of the catatonic phase, while C.S.F. total N had its maximum in the beginning of the catatonic phase.

Total N in serum showed great fluctuations apparently with the same rhythm as the menstruation.

The menstruation was irregular during the first 5 months, with long periods of amenorrhoea: but since then it has been quite regular. Menses occurred at different places in the interval and catatonic phases, having its own rhythm independent of the psychotic phases (see Fig. 1).

INVESTIGATIONS

Urine: The amino acids in the urine were investigated daily during an interval and a catatonic phase, when the patient was not menstruating. She received 4 brewer's yeast tablets (each tablet fortified with 2 mg. thiamine, 2 mg. lacto-flavine and 20 mg. nicotinic acid amide), but no cod liver oil or mineral salts.

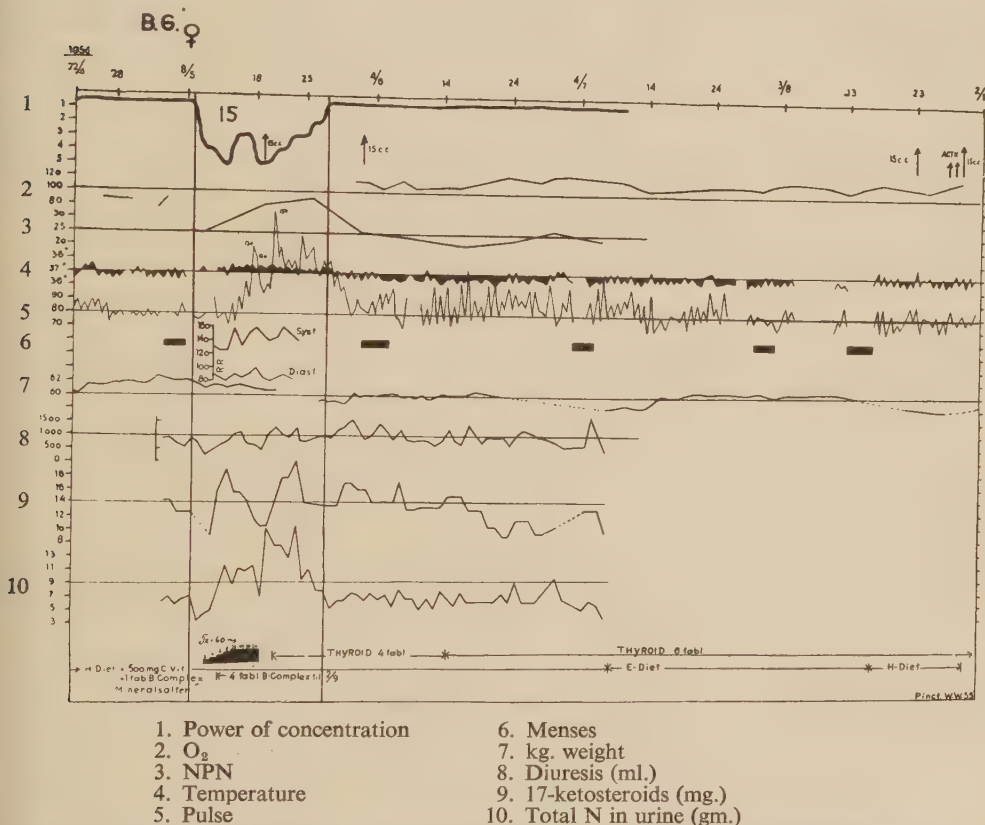


FIG. 5.—This figure demonstrates the action of thyroxine on NPN, temperature, pulse, weight, diuresis, 17-ketosteroids and total N in urine, as well as on BMR.

The thyroxine treatment was started in the beginning of the catatonic phase when NPN was low. The arrows in the upper part show when the patient was tapped for spinal fluid, and when she was given the A.C.T.H. as test.

About a month later the investigation was repeated, but this time the patient was menstruating and she received 8 tablets of brewer's yeast and in addition one tablespoonful of cod liver oil and mineral salts. As Figure 3 shows, the excretion in the urine of amino acids, total nitrogen and 17-ketosteroids, and the pulse and temperature, were considerably modified. The pattern was completely changed, probably because of the menstruation and possibly also because of the increased amount of the B vitamins (?).

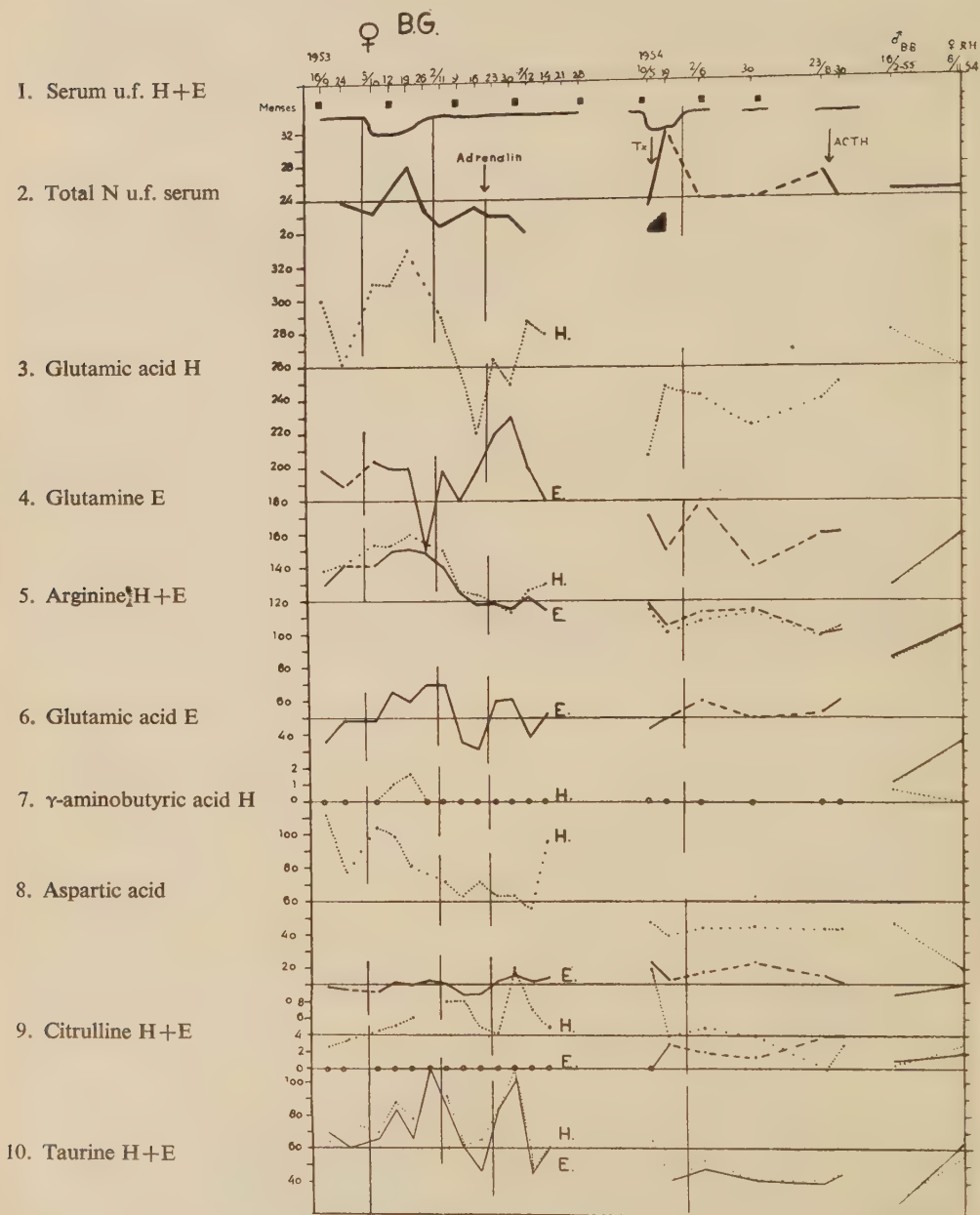


FIG. 6.—Amino acids in ultrafiltered serum.

This figure shows how some amino acids and total N in u.f. serum are changing during the catatonic phase and interval, as well as after adrenaline test, thyroxine treatment and A.C.T.H. test, in the patient compared with one single observation of the two normal controls (♂ B.B. and ♀ R.H.).

The pattern without menses probably gives a picture of the catatonic phase.

Serum and Cerebro-spinal Fluid: Six months later we investigated the amino acids in ultra-filtered serum and C.S.F. and also in native C.S.F. This

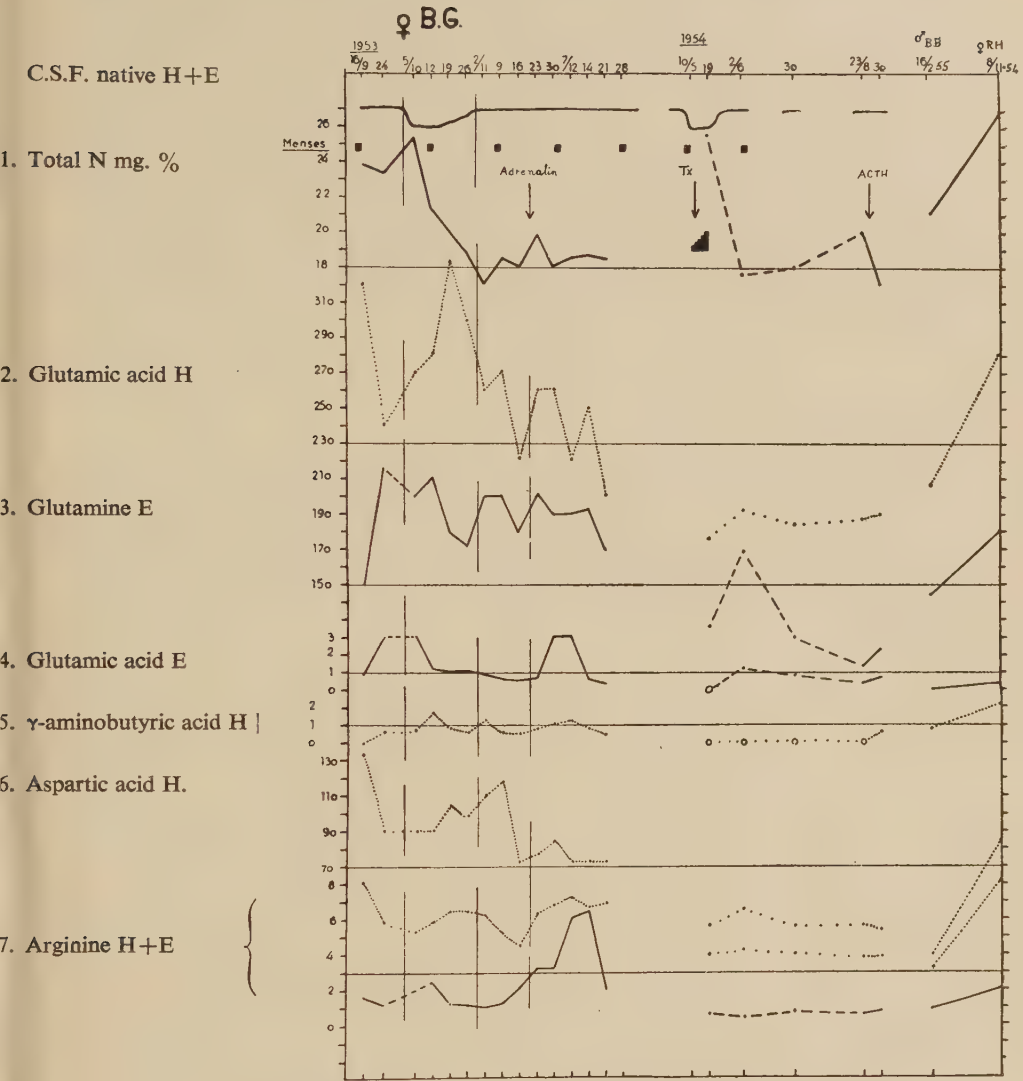


FIG. 7.—Amino acids in native Cerebrospinal fluid.

This figure illustrates the amino acids and total N in C.S.F. under the same circumstances as in Figure 6.

was carried out weekly for about three months, throughout one interval and one catatonic phase and further into the next interval before the next expected catatonic phase.

However, this did not occur for about four months, possibly because of the frequent lumbar punctures (16 ml. C.S.F. weekly) and withdrawal of blood (40 ml. weekly), and/or maybe because of the intravenous injection of adrenaline (0.05 mg. two mornings in succession resulting in a rapid pulse up to 140/min. after 24 seconds. Simultaneously the systolic blood pressure rose from 120 to 220 mm.). The blood pressure investigation was carried out at the time when we expected the next catatonic phase.

In this period of investigation the patient received only 1 tablet of brewer's yeast, 500 mg. vitamin C, cod liver oil and mineral salts (see Fig. 4).

Thyroxine Treatment: When the patient again had regular catatonic phases she was (in May, 1954) treated with 60 mg. thyroxine followed by thyroid substance (6 tablets each containing 0.3 g. of dried thyroid) as described by R. Gjessing (see Fig. 5). The amino acids were investigated during and after the thyroxine treatment in the blood and C.S.F., and immediately before giving thyroxine also in the blood. Since this treatment started, the patient has been well without any psychotic disturbances. She was discharged from the hospital in February, 1955 and since then she has been employed in a full-time job, until June, 1956, when she had an artificial abortion and sterilization, and shortly after she relapsed and was re-admitted to Dikemark, where she had 2 catatonic phases lasting 3 and 6 weeks. Since then she has been free from symptoms.

Normal Controls

Besides the patient, who served as her own normal control, we also investigated the blood and C.S.F. of one healthy male and one female (about the same age as the patient) after they had been living on the same diet as the patient for a week. The excretion of amino acids in the urine was examined in ten psychotic patients and in ten healthy persons. The standard fluid diet and the brewer's yeast tablets were also investigated for their amino acid content.

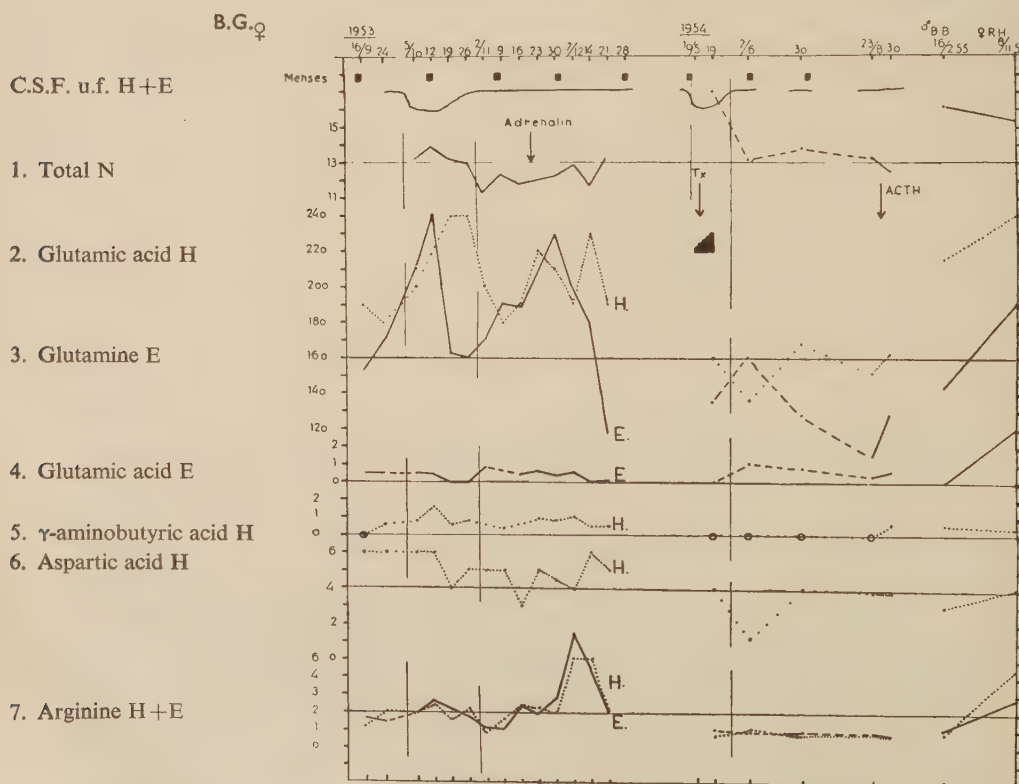


FIG. 8.—Amino acids in ultrafiltered C.S.F. This figure illustrates the amino acids and the total N in u.f. C.S.F. under the same circumstances as in Figure 6.

Our semiquantitative method, which involves estimation of intensity by eye, is not an ideal method, and we only put forward our results as tentative.

RESULTS

In Urine, Serum and C.S.F.

1. We have not found any specific "psychotic" amino acid in the urine, blood or C.S.F. On the other hand, we were unable to find any free citrulline in ultrafiltered serum during the lucid interval or catatonic phase; but this amino acid did occur after thyroxine treatment and it was present in both the normal controls.

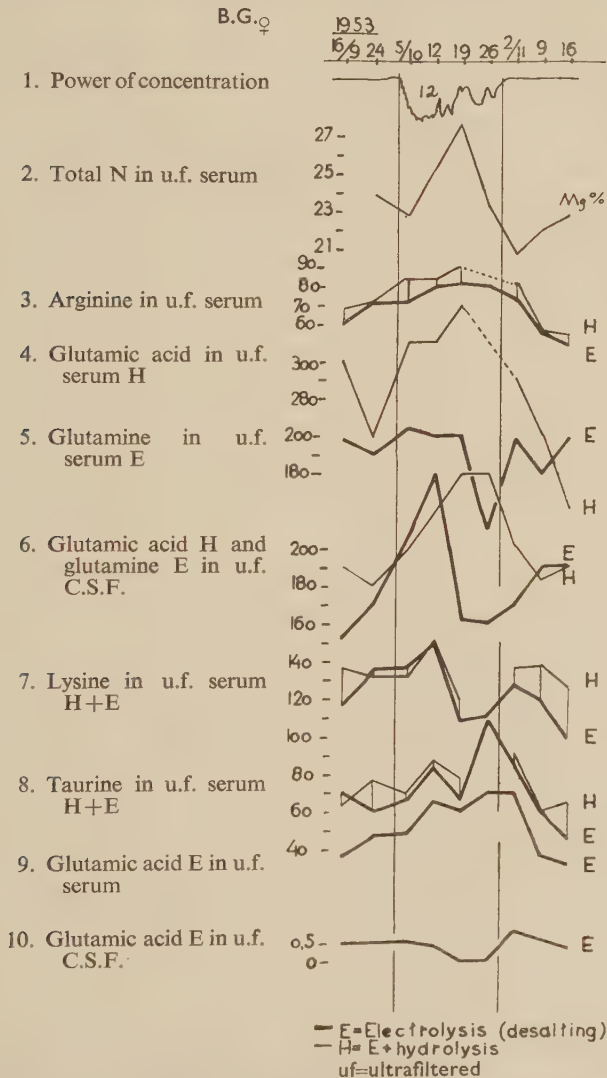


FIG. 9.—This figure is correlating the catatonic phase with the total N in ultrafiltered serum, the arginine in u.f. serum, peptide-bound glutamic acid (H) and free glutamine (E) in u.f. serum and u.f. C.S.F., demonstrating the shift in glutamine-glutamic acid in the middle of the catatonic phase, where arginine and total N in u.f. serum are reaching their maxima, indicating a connection between urea (total N), arginine and glutamine.

Lysine and taurine are probably also operating in the detoxication of ammonia?

2. The most striking observation during the catatonic phase was a shift in the relation between glutamine and glutamic acid: the glutamine reached its maximum level in the first half of the catatonic phase, in the urine and serum and especially in the C.S.F., and reached its minimum at the end of the same phase. On the other hand the glutamic acid reached its maximum when the glutamine reached its minimum level.

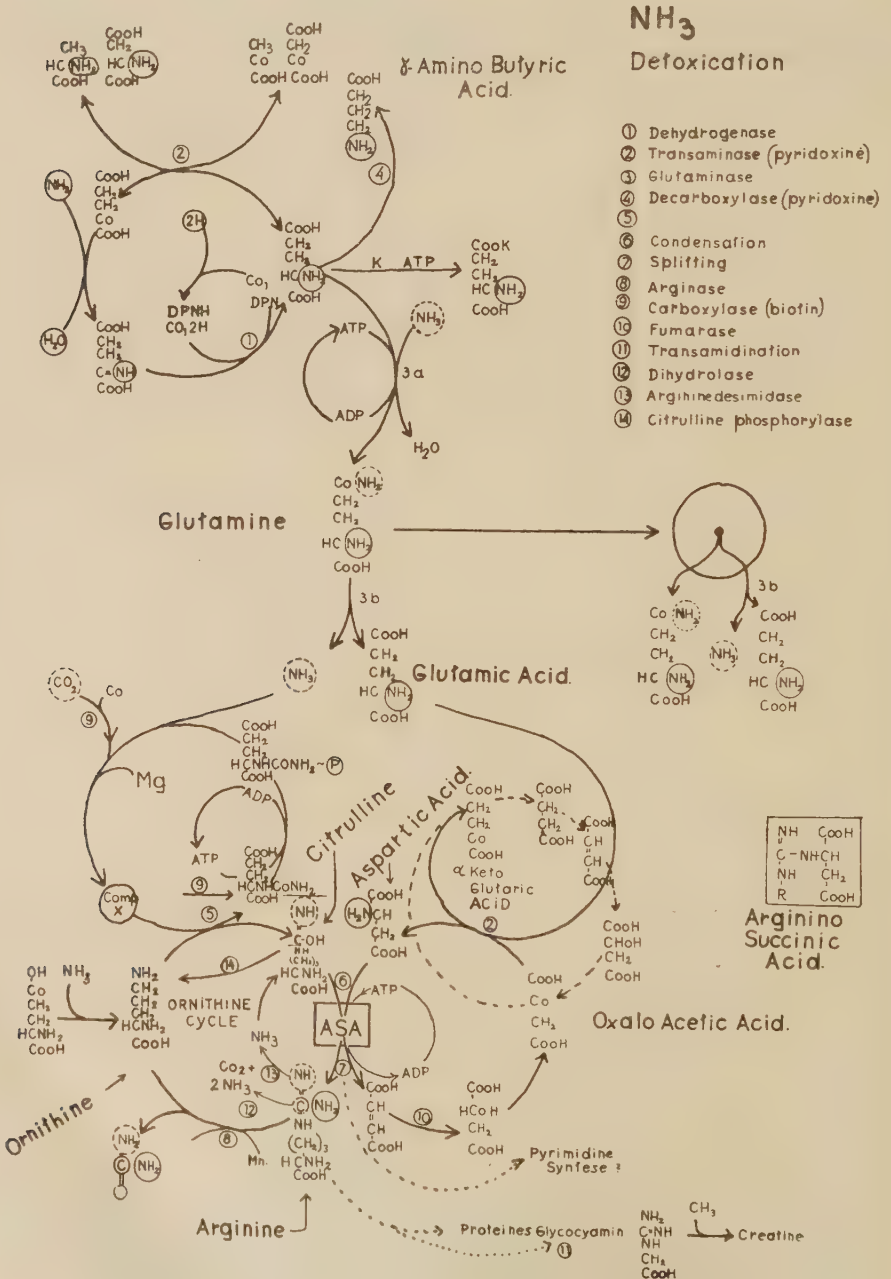


FIG. 10.—Illustrating the process of NH₃ detoxication.

3. Arginine reached its maximum level in the middle of the catatonic phase in the urine and serum.

4. Intravenous adrenaline increased the glutamine and glutamic acid levels in ultrafiltered serum and C.S.F.; taurine increased also in the serum, but arginine did not.

5. Thyroxine treatment seemed to increase the glutamic acid and decrease the glutamine level. Arginine, taurine and peptide-bound aspartic acid decreased and remained at a low level in ultrafiltered serum (see Fig. 6).

6. A.C.T.H. (150 mg.) increased at the beginning of one interval especially the excretion of histidine, taurine, alanine and glycine in the urine.

A.C.T.H. (100 mg.), 3 months after thyroxine treatment, did not influence the amino acids in the serum and C.S.F. very much.

7. On the whole, those amino acids in the serum which had their maximal levels at the beginning or in the middle of the catatonic phase, decreased after treatment with thyroxine (free glutamine, arginine, peptide-bound aspartic acid, free alanine and peptide-bound glycine). The same is true for taurine, peptide-bound citrulline, serine and threonine, even though these four did not have their maxima at the same place. On the other hand those amino acids which had their maxima towards the end of the catatonic phase, increased after thyroxine (peptide-bound glutamic acid, leucine, valine, tyrosine, phenylalanine, and alpha-aminobutyric acid).

8. As can be seen in Figure 4, the total N in the serum varied significantly, with a minimum level premenstrually and a maximum between the menses, independent of the psychotic phases.

9. The most important observation was probably the change in the pattern of amino acids excreted in the urine, with and without menstruation (see Fig. 3).

DISCUSSION

The periodic changing in the excretion of urea in the urine may be due to a periodic deficiency of arginase and glutaminase. Our findings of a relative increase of arginine and glutamine in the urine, serum and C.S.F. in the first part of the catatonic phase seem to point to this possibility (see Figs. 9 and 10). Insufficient arginase and glutaminase could probably produce a slight ammonia intoxication that could influence the central nervous system in a psychotic direction.

In the latter half of the catatonic phase the arginase is apparently activated (if the amino acids in the serum really give a picture of the conditions in the liver), since the urea excretion in the urine increases at the same time as the arginine and glutamine decrease in the blood and urine.

Thyroxine and thyroid substance in large doses compensate the condition (by activating autonomic centres in the diencephalon?), and they seem to prevent any specific disturbance of urea synthesis.

From another viewpoint, all our findings may just illustrate an unspecific metabolic reaction or response to an endogenous intoxication in a patient with insufficient thyroid function.

SUMMARY

Twenty-five amino acids were investigated by paper chromatography, semiquantitatively, daily in the urine for three months, and weekly in ultrafiltered serum and C.S.F., as well as in native C.S.F. for three months, both with and without hydrolysis. We investigated only one

patient suffering from periodic catatonia. The investigation was carried out throughout the catatonic phase and the lucid interval, before, during, and after compensation with thyroxine and dried thyroid substance. The patient as well as the normal controls were kept on a standard fluid diet.

The results are based on a semiquantitative method with many pitfalls and with an error of ± 10 –15 per cent. Therefore the results are only tentative and by no means conclusive.

The hypothesis of a deficiency of arginase and glutaminase is put forward as an explanation of the variations in arginine, glutamine and glutamic acid. Thyroxine compensates the condition, probably by activating autonomic centres in the hypothalamus, and possibly by preventing specific disturbances of urea synthesis.

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COMBINED CHLORPROMAZINE AND RESERPINE IN THE TREATMENT OF CHRONIC PSYCHOTICS

By

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In the past few years many papers have appeared both in Europe and the United States of America demonstrating the value of Chlorpromazine and Reserpine in the treatment of mental illness. In the United States of America Kinross-Wright (1954) has reported on Chlorpromazine in the treatment of schizophrenia and claimed very satisfactory results; in this country Lomas (1955) and his collaborators have in several papers confirmed these results. With regard to Reserpine in the United States of America Kline (1954) has used the drug extensively with good results; in Europe, hopeful reports by Foote (1955) and McGrath *et al.* (1956) confirmed the value of this drug in psychiatric practice.

Interest in using these drugs in combination naturally followed and papers on such use have appeared. Hollister *et al.* (1955) dealing with 32 cases of chronic schizophrenia concluded that the combination held no advantage over the use of either drug alone. Hormia (1956) however, was of the opinion that the combination was much more effective. Barsa and Kline (1955) report the combined drugs to be more effective than Reserpine alone, but claim the addition of Chlorpromazine resulted in the occurrence of jaundice and agranulocytosis.

When, therefore, the opportunity to try this combination with the drugs combined in one tablet presented itself, it was decided to try the drugs on a group of patients who, at that time, were considered to have a poor prognosis.

METHOD

Seventy female patients were selected for this trial in all of whom the prognosis was considered to be poor. Behaviour problems were present in nearly all and many were only kept on a reasonable level by means of regular maintenance E.C.T.

The group consisted of 53 schizophrenics, 4 mentally backward patients with schizophrenia superimposed, 9 with manic-depressive-psychosis, 2 epileptics and 2 psychopathic personalities.

Twenty-four of these patients had received Chlorpromazine previously with only transient or no improvement, 7 had had Reserpine, 20 had received courses of Deep Insulin and 19 had undergone prefrontal leucotomy.

The average length of continuous stay in this hospital was $8\frac{1}{2}$ years, but many had had previous admissions to this and other mental hospitals and all but 11 had been here for more than 3 years.

Forty-five of the patients received the active drugs and 25 received control tablets of identical appearance. Only the hospital pharmacist knew which patients were receiving the active drugs or the placebo, the choice having been made by him at random.

The patients were all nursed in the same ward. The nursing staff were increased in number so that full observation might be obtained. Occupational Therapy classes, both in the Therapy Villas and on the ward, were organized, and reports were received on the patients' progress in this department from the therapists. Full physical examinations were carried out prior to starting treatment, as was also a clinical assessment of the patients' mental status.

Throughout the five-month trial daily blood-pressure recordings, twice daily T.P.R. readings and weekly weights were recorded but no significant findings were noted.

The drugs were initially supplied in chocolate-covered tablets containing Chlorpromazine 50 mg., Reserpine 0.5 mg., and at first the patients were given 1 tablet three times a day by mouth. This dose, however, was found to produce side-effects of a Parkinsonian nature in many patients, and adjustment of the dose to prevent this was difficult. However, the manufacture of tablets of half strength solved this difficulty and enabled the dose to be adjusted satisfactorily.

RESULTS

In 3 cases the trial was discontinued; one because the relatives objected to the drowsy state of the patient noted by them when visiting, one, a psychopath, who was a voluntary patient, discharged herself against advice, and the third, a patient who developed status epilepticus, after the occurrence of which it was felt wise to discontinue the drugs.

The assessment of the remainder was made at the end of the 5-month period of the trial and prior to the scrutiny of the pharmacist's list of control patients.

The assessment was made on the following lines:

- A. Symptom free—insight good—affect normal.
- B. Symptom free—fair insight—affect flattened.
- C. Behavioural improvement marked—symptoms less evident and often only elicited on questioning.
- D. No improvement.

11 cases were placed in Category A.

8 cases were placed in Category B.

11 cases were placed in Category C.

37 cases were placed in Category D.

On checking this list with the pharmacist's list it was found that 2 of the Category A patients were controls, the diagnosis being manic depressive psychosis in one case, schizophrenia superimposed on a limited intelligence in the other. In Category B all patients were on the active drugs whilst in Category C, 2 patients had received the dummy tablets. Both of these patients were schizophrenics, one, a Pole, could hardly speak any English and behaviour was the only yardstick for judging her improvement. Among the cases in Category C was a case of epilepsy who, prior to treatment, kept up a constant stream of obscenities, usually sung in tune to a well-known hymn. All anti-convulsant drugs were stopped in this case and the incidence of her fits was not increased. The patient improved considerably, lost her obscene habits, became co-operative in every way.

In view of the epileptic side-effects mentioned later, it is interesting that no increase in fits occurred in this case.

A few weeks after the start of the trial the whole atmosphere of the ward changed. From being the usual disturbed ward with periodic outbursts of noisy violence, an air of tranquillity prevailed. One patient, who was extremely violent and aggressive, improved to such an extent that complete dental examination and treatment was carried out whereas before it was impossible to get her near the dentist's chair. This patient, and one other who was considered to be impulsively suicidal and a potential escapee, at first appeared to be making excellent remissions but these were unfortunately not fully maintained and whilst both are improved in the final assessment they were placed in Category C.

The increased attendance in the Occupational Classes was marked and whilst some of the controls also attended, the main increase was from the treated patients. Willingness to assist in ward work was shown by many patients who previously would do none. Many patients attended dances, the cinema, and other recreational activities to which they were unable to go before.

Of the 11 patients in Category A, 10 have been discharged, including the two who were controls. These went home on trial at first and without drugs.

In one of the discharged cases only has there been any setback, and this quickly settled when the patient was put back on the Chlorpromazine/Reserpine mixture. Several of the Category B patients have been regraded from Certified to Voluntary Status and efforts are being made to find them jobs and accommodation.

Of the 45 patients who received the active drugs, 42 completed the course, and of these 21 per cent. were in Category A; 19 per cent. Category B, and 21 per cent. in Category C; 61 per cent. therefore of the treated patients showed varying degrees of improvement.

SIDE EFFECTS

The chief side-effects noted were:

1. *Parkinsonism*

Eight patients at various times during the trial showed sialorrhoea, 7 showed tremor, 5 showed the typical facies, 2 had oculogyric crises and 1 paralysis of accommodation.

Twelve patients developed an oily seborrhoeic condition of the skin affecting the hair of the head and the face.

2. *Fits*

Four patients, all of whom had previously undergone leucotomy, had one or more epileptic fit during the treatment. One patient, who had not been leucotomized, a high-grade defective who has been in this hospital since the age of 13 and in whom there was no history of epilepsy, developed status epilepticus.

3. *Drowsiness*

Twelve patients at one stage or another complained of this symptom but in all cases it was controlled successfully by the use of Amphetamine Sulphate.

4. *Oedema*

Four cases showed oedema of both ankles and face. A day or two without the drug brought about a subsidence of this.

5. Hypotension

Two cases showed transient hypotension in the early stage of the trial but this settled later.

In considering these side effects it is of interest to note that whereas Parkinsonian symptoms have been described by many authorities, these have normally followed much larger doses. Kinross-Wright (1955) claims it rarely occurred on less than 200 mg. of Chlorpromazine daily whereas several of these patients showed it on considerably smaller doses. It appears, therefore, that the two drugs in combination have potentiated each other with regard to this. Hormia (1956) however, reports his findings that the drugs in combination provide less side effects.

With regard to the fits, the occurrence here was in keeping with the findings of other trials, namely, that the incidence was mainly in individuals who had previously had leucotomy. Lomas *et al.* (1955) report 10 cases in 6 of whom prefrontal leucotomy had been performed. They suggest that Chlorpromazine has epileptogenic properties in susceptible people.

Oedema has been reported with Reserpine (Foote, 1955); and in view of the fact that hypotension occurs with both drugs the incidence in combination is satisfactorily small.

DISCUSSION

The results obtained in a group of cases consisting largely of poor material is very satisfactory. This combination of Chlorpromazine and Reserpine seems to have been more successful than either drug used alone, but the side effects require careful attention and dose variation.

It is of interest that four patients receiving the control tablets improved and this is possibly explained by the increased interest and activity in the ward resultant upon the trial taking place, plus the additional occupational therapy facilities, etc., arranged.

The results of this trial suggest that the combined drugs have a real place in the treatment of the difficult anti-social chronic psychotics and judging by the results so far obtained it appears that if their remission is good the majority of patients so treated do not need to remain on the drugs after discharge, and if relapse occurs, may respond again to further treatment with the same combination.

SUMMARY

Seventy anti-social female patients were nursed in the same ward, 45 received combined Chlorpromazine/Reserpine, 25 received Control Tablets, the choice being known only to the pharmacist.

Side effects mainly of a Parkinsonian type occurred fairly frequently.

The degree of remission obtained in such unpromising material was satisfactory.

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A COMPARISON BETWEEN THE RESULTS OF UNMODIFIED AND MODIFIED ELECTROPLEXY (E.C.T.)

By

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SINCE the discovery of the therapeutic value of induced epileptic convulsions in mental illness, there have been efforts to make this form of treatment available to those patients whose psychiatric state would benefit from it but whose physical condition contraindicates its use. Modifications have been sought, particularly since the introduction of electrical methods of inducing the fit. The modification of electroplexy takes three main forms—postural, electrical and chemical (Montagu, 1953) and it is with this third type, the use of anaesthetic and relaxant drugs, that this paper is concerned.

The reasons for modification of the convulsion are firstly to enable the treatment to be given to those patients whose physical conditions would otherwise preclude it; curare was the first relaxant drug used by Bennett (1940) to prevent traumatic complications and it has enabled electroplexy to be given to patients with advanced cardiac disease, arthritic deformities and other severe physical illness; and secondly to prevent such complications as skeletal injury. With the introduction of the short-acting relaxants the safety of the method was increased and the use of modified electroplexy became more frequent so that in many hospitals it is now given routinely (Lincoln and Broggi, 1955).

In a recent court case (*Bolam v. Friern Hospital Management Committee*—*Times* Law Report, 1957) the plaintiff alleged that failure to use relaxant drugs was negligent treatment. The Judge, Mr. Justice McNair, stated that a professional man was not guilty of negligence if he acted in accordance with a practice which was accepted by a competent body of professional men skilled in that particular art, but he also mentioned that the jury must not look with 1957 spectacles at what had happened in 1954, suggesting the possibility that failure to use relaxants might now be considered negligent.

Montagu points out that since it is impossible to predict accurately which of the healthy subjects are going to develop complications during unmodified treatment, it follows that any successful method of preventing these catastrophes should be used as a routine, always provided that the balance of risks is in favour of the method rather than of the complications it is designed to prevent. As far as the merits of the method itself are concerned there are two opposing views exemplified by Bennett (1943) at one extreme who says that straight convulsive shock is contraindicated, and more recently Kelleher and Whiteley (1955) who say it is difficult to justify the use of electroconvulsive therapy without muscle relaxants; while Kalinowsky (1949) feels that convulsions are a preformed mechanism which can be safely elicited in any individual while curare is more dangerous than the complications it is supposed to prevent. Whether his views are the same about the shorter-acting relaxants is not known,

but with these, which are much safer than curare even in comparatively inexperienced hands, the question of the results of treatment has also to be included in any consideration of its indications. Maclay (1953) took a serious view of the position concerning the use of relaxants, though this was before the general use of the short-acting group, and suggested that they should be used with caution.

While there have been several papers describing different techniques of administration of relaxant drugs, with or without an anaesthetic (Rietman and Delgado, 1955; Gillie and McNeill, 1955; Kelleher, 1955; Stevens *et al.*, 1954) there have been few references to results obtained compared with unmodified treatment. Fisher and Bannister (1953) and Lincoln and Broggi (1955) mention clinical impressions, without quoting figures, that the psychiatric results of modified E.C.T. are at least as good as those of straight E.C.T. and that patients have not complained about unpleasant sensations and show less tendency to develop apprehension during the course of treatment when modified. Ardis and Wyllie (1953) note that while in general modified E.C.T. has been as effective therapeutically as unmodified E.C.T., in a few cases this was apparently not so. Holmberg *et al.* (1956) found in endogenous depressions that the therapeutic effect tended to be better when muscular relaxation had been employed, and that a smaller number of treatments were given. The difference was not statistically significant, either in respect of therapeutic improvement or in number of shocks given. Rietman and Delgado note that the average number of treatments given is the same whether modified or unmodified and they comment that although vastly reducing the usual complications the relaxant did not inhibit the therapeutic effectiveness of the treatments. However, their use of the word "unmodified" implies only the lack of relaxant, because they mention that light anaesthesia with thiopentone was induced before the shock was administered in their "unmodified" patients.

In contradistinction to these views, it has been the clinical impression at this hospital, both among the medical and nursing staff, that some patients seemed to respond poorly to modified electroplexy, as noted also by Ardis and Wyllie. When a patient had a poorly modified fit due to faulty injection or a similar reason, a typical nursing comment would be "Oh, she's had a good fit, she'll be better now". At first this was felt to show an unawareness of the purpose of modification but enquiry showed that it was the opinion of both experienced nursing staff and doctors that some patients whom they would have expected to respond to electroplexy did not in fact do so when a relaxant was used, or else responded less well. The medical staff also noticed that an occasional patient did not respond to a full course of modified electroplexy but after a short rest period reacted quickly to a course of unmodified treatment. This was first thought to be due to a repetition of treatment, particularly as it was given later in the illness, but a succession of similar experiences suggested the need for investigation of the facts. It was decided to compare the results of modified with unmodified treatment given at this hospital since 1 January, 1954.

METHOD

The first step has been the retrospective examination of the records of female patients discharged from this hospital since 1 January, 1954; this date was chosen because since that date both types of treatment have been regularly used in the hospital, and a considerable number of the treatments have

been given either by myself or by doctors trained in the same technique, under my supervision. It was unfortunate that a similar examination of the records of male patients was not possible but the filing of treatment records was carried out in a different way and not readily available for this survey. Two Electroplexy machines were in use, the Macphail-Strauss Plexacon and the Ectron.

UNMODIFIED ELECTROPLEXY

The patient lies on a firm bed or an examination couch, a nurse on either side supporting the shoulders and pelvic girdle, a rubber gag is placed in the mouth by the Sister who then applies the electrodes to the temples. A diphasic shock of 20 Joules from the Plexacon or one second from the Ectron is given. After the fit the patient is turned on her side and oxygen administered for the first few breaths. Atropine premedication is not given routinely.

MODIFIED ELECTROPLEXY

0.2 Gm. thiopentone in 5 per cent. solution mixed with 40–60 mg. suxethonium bromide in 2 per cent. solution or 2.5 mg. decamethonium iodide is used, the dose of thiopentone being constant except in very obese or very small patients and the relaxant dose being estimated by the size of the patient but without accurate weighing, sufficient being given to obtain a considerable degree of relaxation but yet to be sure that a fit has taken place. The solutions are mixed in the same syringe just before administration. After an initial 1 ml. followed by a pause and questioning for pain to see if there is evidence of intra-arterial injection of the thiopentone the injection is completed as rapidly as possible. No premedication is given. The shock, 25 Joules with the Plexacon or 2 seconds with the Ectron, is given as soon as the muscular twitchings have subsided. The jaw is held shut by the Sister, no gag being used, while the doctor gives the injection and then operates the machine. It is not considered necessary to have a qualified anaesthetist present provided that the psychiatrist has knowledge of the principles involved in dealing with an unconscious patient (Mushin, 1955); this applies as much to unmodified as to modified electroplexy. The patient is turned on her side and oxygen is administered by controlled intermittent artificial respiration until spontaneous breathing returns—in a number of timed cases this took from $2\frac{1}{2}$ to 5 minutes from the beginning of the injection to the first satisfactory breath.

Modified treatment was given when there was a definite indication, such as a fracture, but when other patients were referred for electroplexy the method of treatment depended more on the views of the registrar giving the treatment concerning the advantages and disadvantages of the straight and modified types, than on the ideas of the person referring the patient for treatment. This was because there was considerable argument about the value of routine modified electroplexy. Therefore, it is possible to assess the results of the two types of treatment with the likelihood that each group contains a representative sample of all the patients receiving treatment during the period. There would be a tendency for patients who were severely ill to be included in the modified electroplexy group, and where there was an obvious severe physical illness noted this case was excluded from the series since the factors examined, length of stay in hospital and degree of recovery, may be altered by the presence of physical illness. Only cases diagnosed on discharge as Manic-depressive and Recurrent Depression, Involutional Depression, Neurotic Depression and Puerperal Depression were examined. If cases of schizophrenia were included

it was felt that too many complicating factors were likely to be present, such as concomitant Deep Insulin treatment.

RESULTS

The records were examined from two points of view. Firstly the immediate effects of treatment were assessed and to estimate these the following points were considered:

- (a) Length of stay in hospital.
- (b) Number of treatments given.
- (c) Number of refusals of treatment.
- (d) Gain or loss in weight.
- (e) Number of patients discharged "recovered" compared with those discharged "relieved".

Secondly a follow-up survey of all those patients who had been discharged for at least a year was carried out.

Immediate Effects of Treatment

A total of 438 records were examined but since each period of admission if separated by more than one month from the previous date of discharge was counted, there were in all 472 admissions, with 26 patients having two and 4 having three admissions during the period under review. Each of these 472 admissions will be dealt with as a separate case when dealing with the immediate results of treatment. In the four diagnostic groups considered there were 172 cases of manic-depressive psychosis, usually recurrent depressions, 89 of involutional depression, 192 of neurotic depression and 19 of puerperal depression. These diagnostic categories are based on standard psychiatric practice as described in Henderson and Gillespie's *Textbook of Psychiatry* (1956). Of the 472 cases, 328 received unmodified electroplexy and 144 received the modified form of treatment (Table I).

TABLE I
Numbers of Patients Receiving Unmodified or Modified Electroplexy

Diagnosis	Unmodified E.C.T.	Modified E.C.T.
Manic-depressive psychosis	121	51
Involutional depression	59	30
Neurotic depression	137	55
Puerperal depression	11	8
Total	328	144

Length of Stay in Hospital

The average length of stay for all cases who received unmodified treatment is 57.2 days while for those receiving modified electroplexy it is 71.7 days. This difference is maintained in the various diagnostic groups considered separately. In the manic depressive group the unmodified treatment cases were in hospital for an average stay of 65.5 days, the modified treatment group for 69.4 days; in the involutional depressives the unmodified group were in hospital 50.7 days, the modified group for 62.3 days; the neurotic depressive group for

51.9 and 74.4 days respectively and the puerperal depression group 57.3 and 98.0 days respectively. These results are summarized in Table II.

TABLE II
Average Length of Stay in Hospital
Treatment

Diagnosis	Treatment		t-test of Significance
	Unmodified E.C.T. Days	Modified E.C.T. Days	
Manic-depressive psychosis ..	65.5	69.4	Not significant
Involuntional depression ..	50.7	62.3	Significance at 0.2 level of confidence.
Neurotic depression	51.9	74.4	Highly significant at 0.01 level of confidence.
Puerperal depression	57.3	98.0	Not significant.
All patients	57.2	71.7	Not significant.

In these results, two patients with puerperal depression who received modified electroplexy were in hospital an exceptionally long time, one for 344 days and the other for 214 days, while the total number of cases was 8, so that a false impression is obtained of a marked difference in the average length of stay in hospital when receiving the modified treatment.

This must be borne in mind as a fault of this method of assessment, though it does not necessarily invalidate conclusions drawn from the results. If these two cases are excluded the average stay in hospital for the seven cases of puerperal depression receiving the modified form of treatment was 42.1 days.

Number of Treatments

The average number of treatments given to the unmodified group was 6.2 while the average number of treatments to the modified group was 7.3. In Table III the separate diagnostic groups are shown, and in each case less treatments are required when electroplexy is given unmodified. The manic depressive group require 6.8 as against 7.2 treatments, the involuntional depressions 6.1 as against 7.2, neurotic depressions 5.8 as against 6.0 and the puerperal depressions 6.0 against 9.1 (Table III).

TABLE III
Average Number of Treatments Received

Diagnosis	Treatment	
	Unmodified E.C.T.	Modified E.C.T.
Average No. of Treatments		
Manic-depressive psychosis	6.8	7.2
Involuntional depression	6.1	7.2
Neurotic depression	5.8	6.0
Puerperal depression	6.0	9.1
Total	6.2	7.3

t-test shows that the difference between the means is not statistically significant.

Number of Refusals of Treatment

Included under this heading are all those patients who refused to continue the course of treatment at any stage once they had received the first treatment.

Also included are those who refused at some time during the treatment but who later agreed to continue it. If a patient refused to receive treatment on more than one occasion during a single admission this is counted as one refusal, but if on different admissions, each course of treatment is dealt with independently of the previous ones.

Fourteen patients refused unmodified treatment and 8 refused modified treatment. The ratio of the number of unmodified to modified treatments is 328 : 144 which is 2.28 : 1, while the ratio of refusals 14 : 8 is 1.75 : 1. This result is not significant statistically—thus there is no undue dislike as indicated by refusal of treatment, for unmodified electroplexy.

Gain or Loss of Weight

Following the findings of Altschule *et al.* (1948) that those patients who gained weight after E.C.T. were improved while those who failed to gain showed no striking clinical improvement, the change in weight of all patients during the period in hospital was noted. The average weight gained in the patients receiving unmodified treatment was 2.6 lbs. while in modified treatment the average gain was 1.3 lbs. A t-test showed there was a significant difference between these means at the 0.02 level of probability.

There was thus a significantly greater average gain in weight in those patients receiving unmodified rather than modified treatment. Altschule attributed the gain in weight to retention of extracellular fluid and found that it ushered in clinical recovery in the large majority of cases.

To see if a gain in weight may be considered a good prognostic sign and loss of weight a poor one the results were examined to see what had happened to those patients who gained or lost weight. Of those who gained weight, 210 (63.1 per cent.) were discharged recovered and 123 (36.9 per cent.) were discharged relieved. Of those who lost weight, 87 (66.9 per cent.) were discharged recovered and 43 (33.1 per cent.) relieved. When seen after a year, of those patients who had gained weight in hospital, 202 (75.6 per cent.) remained well, 60 (22.5 per cent.) had relapsed and 5 (1.9 per cent.) died. Of those who lost weight, 71 (71 per cent.) remained well, 27 (27 per cent.) relapsed and 2 (2 per cent.) died. Chi-square tests of significance carried out on these figures did not reach a satisfactory level of significance—thus the gain in weight while in hospital is not a satisfactory guide to prognosis in this series (Table IV).

TABLE IV
State of Patients Who Gained or Lost Weight

	State on Discharge		Follow Up		
	Relieved	Recovered	Remained Well	Relapsed	Dead
Gain in weight in hospital	210 (63.1%)	123 (36.9%)	202 (75.6%)	60 (22.5%)	5 (1.9%)
Loss of weight in hospital	87 (66.9%)	43 (33.1%)	71 (71%)	27 (27%)	2 (2%)

State on Discharge

The last point to be considered in the condition of the patients at the time of their stay in hospital is the condition on discharge. The standard nomenclature of "Recovered", "Relieved" and "Not Improved" has been used and the opinion of the clinician seeing the patient on discharge is that which has been noted. In the Manic Depressive group receiving unmodified treatment,

64 (52.9 per cent.) were discharged Recovered and 57 (47.1 per cent.) Relieved. In the modified group, 22 (45.0 per cent.) were discharged Recovered and 29 (59.2 per cent.) Relieved. In the Involutional Depression group receiving unmodified treatment, 23 (47.0 per cent.) were discharged Recovered, 24 (49.0 per cent.) Relieved and 2 (4.1 per cent.) Not Improved; while for those receiving modified treatment 12 (40.0 per cent.) were Recovered, 17 (56.7 per cent.) Relieved and 1 (3.3 per cent.) Not Improved. In the Neurotic Depression group receiving unmodified treatment, 35 (25.6 per cent.) patients were discharged Recovered, 97 (70.8 per cent.) Relieved and 5 (3.7 per cent.) Not Improved. Those receiving modified treatment, 6 (11.0 per cent.) were discharged Recovered and 49 (89.1 per cent.) Relieved. In the small group of Puerperal Depressions receiving unmodified treatment, 6 (54.6 per cent.) were discharged Recovered and 5 (45.4 per cent.) Relieved and of those receiving modified treatment, 4 (50 per cent.) were discharged Recovered, 4 (50 per cent.) Relieved.

These results are summarized in Table V; (both in actual numbers of patients and in percentages of the total number of patients receiving the particular form of treatment).

TABLE V
Condition of Patients on Discharge

Diagnosis	Unmodified E.C.T.			Modified E.C.T.		
	Recovered	Relieved	Not Improved	Recovered	Relieved	Not Improved
Manic-depressive psychosis	64 52.9%	57 47.1%	0 0%	22 45.0%	29 59.2%	0 0%
Involutional depression ..	23 47.0%	24 49.0%	2 4.1%	12 40.0%	17 56.7%	1 3.3%
Neurotic depression ..	35 25.6%	97 70.8%	5 3.7%	6 11.0%	49 89.1%	0 0%
Puerperal depression ..	6 54.6%	5 45.4%	0 0%	4 50.0%	4 50.0%	0 0%

The assessment of results was also carried out using age rather than diagnosis as a criterion and a similar series of results were obtained (Table VI).

TABLE VI
Relationship Between Age and Unmodified and Modified E.C.T.

Unmodified E.C.T.

Age		Stay in Hospital (Days)	Average No. of Treatments	State on Discharge		
				Recovered	Relieved	Not Improved
Less than 40	..	53.8	6.2	39 32.0%	81 66.4%	2 1.6%
40 to 60	..	57.8	6.2	72 42.6%	94 55.6%	3 1.8%
Over 60	..	57.8	6.2	15 40.6%	20 54.1%	2 5.3%

Modified E.C.T.

Age		Stay in Hospital (Days)	Average No. of Treatments	State on Discharge		
				Recovered	Relieved	Not Improved
Less than 40	..	80.9	7.6	5 15.2%	28 84.9%	0 0%
40 to 60	..	73.4	6.5	22 31.9%	47 68.1%	0 0%
Over 60	..	65.0	6.4	18 42.9%	23 54.8%	1 2.3%

Complications of E.C.T.

The commonest complication of unmodified electroplexy is the occurrence of fractures either of the vertebrae or of the long bones, or dislocation of joints. In a review of the problem, Newbury and Etter (1955) point out the difficulties of diagnosis without X-ray before and after treatment and state that clinical examination is not a reliable guide nor is subjective pain a satisfactory indicator. They give an incidence of 0·5 to 35·4 per cent. of fractured vertebrae occurring after unmodified electroplexy in their review of the literature, the variation depending on when X-rays were taken and whether diagnosis was made by a radiologist.

In this series 5 patients were X-rayed after complaining of severe back pain following treatment. Fractures were found in each case, four having mid-thoracic vertebrae fractured and one with the second lumbar vertebra fractured. The fractures were noted after 1, 4, 3, 3 and 2 treatments. Only two of these patients were included in the follow-up survey and neither complained of difficulties resulting from the fractures. Both agreed they would have treatment again if it were indicated.

There were no deaths which were in any way attributable to E.C.T. in this series. Only one patient died while in hospital, of mesenteric thrombosis.

In the modified group, several patients had relatively prolonged apnoea, two of 15 minutes, but no explanation of this was discovered—serum pseudocholinesterase levels were within normal limits. It is possible that of over-enthusiastic ventilation with its consequent removal of CO₂ may be responsible for the occasional prolonged apnoea and must be guarded against by using intermittent ventilation. It was commoner in the earlier days before experience was gained of the optimum amount of artificial respiration.

In three patients there was noticed a transient cyanosis of the fingers of the hand after the injection had been given. This did not occur immediately but was noted after treatment. In one patient it was limited to two fingers, the third and fourth, and in the other two all the fingers, but not proximal to them. The pulse was good, there was no pain, nor anaesthesia, and the condition cleared spontaneously after about an hour. It was considered due to spasm of the digital veins, resulting in slowing of blood flow and consequent cyanosis, and was thought to be due to the thiopentone rather than the relaxant because in the three patients in whom it occurred one was receiving suxethonium bromide and thiopentone, the second decamethonium iodide and thiopentone and the third thiopentone alone.

Follow-Up of Patients

The second stage of the enquiry was the interviewing of those patients who had been discharged from hospital during the period 1 January, 1954 to 31 December, 1955, and had during their stay in hospital been diagnosed as suffering from one of the four illnesses mentioned and had received electroplexy. There was thus a follow-up period available of at least one year when the survey was carried out in February and March, 1957. The interviewing was done by the Psychiatric Social Workers at this hospital to whom I must pay tribute for the exceptionally good result. There were 388 patients to see and they were able to trace all but 32 (8·2 per cent.). It is considered that this high follow-up rate after one to two years is possible because of the static nature and ready accessibility of small provincial communities compared with the bigger cities and this advantage should be borne in mind when planning any long-term

research. Blair and his co-workers (1957) achieved a similar high follow-up rate (12 per cent. untraced), in the study of out-patient psychotherapy in Plymouth as compared with Mason (1956) in a London hospital where 20 per cent. were untraced and a further 18 per cent. replied by letter only.

The interviewer was asked to assess the patient's condition since discharge and place in one of the following categories:

1. Symptom free.
2. Minor symptoms only. Able to carry on with her usual occupation. Requiring occasional hypnotic or mild sedative only.
3. Definite relapse or recurrence requiring further treatment either as an in-patient or out-patient.
4. Deaths. Tactful enquiry was made to ascertain if the death was due to suicide—not necessarily the verdict of an inquest.
5. No information available.

RESULTS

Three hundred and eighty-eight patients had been discharged from hospital for at least one year at the time of the survey. Information was obtained concerning 356 (91·8 per cent.). Of these 274 had been out of hospital for eighteen months and 206 for two years. These figures, and their division into those who received unmodified and modified treatment are shown in Table VII.

TABLE VII
Number of Patients Interviewed

Period Elapsed Since Discharge	Number of Patients Interviewed		
	Unmodified E.C.T.	Modified E.C.T.	Total
1 year	252	104	356
18 months	199	75	274
2 years	151	55	206

Considering the patients followed up for one year; in the unmodified group, 117 (46·4 per cent.) remained symptom free, 104 (41·3 per cent.) had minor symptoms, 25 (9·9 per cent.) relapsed, 3 (2·0 per cent.) committed suicide and a further 3 (2·0 per cent.) died due to some non-psychiatric illness. In the modified group, 43 (41·4 per cent.) remained symptom free, 45 (43·3 per cent.) had minor symptoms, 13 (12·5 per cent.) relapsed, 2 (1·9 per cent.) committed suicide and one (0·9 per cent.) died of a non-psychiatric cause. For those followed up for eighteen months the figures are as follows: in the unmodified group, 106 (53·3 per cent.) remained well, 74 (37·2 per cent.) had minor symptoms, and 19 (9·6 per cent.) relapsed; in the modified group 36, (48·0 per cent.) remained well, 32 (42·7 per cent.) had minor symptoms, 6 (8·0 per cent.) relapsed and 1 (1·3 per cent.) committed suicide. The two-year follow-up gave the following results: in the unmodified group, 86 (57·0 per cent.) remained well, 51 (33·8 per cent.) had minor symptoms, 12 (8·0 per cent.) relapsed, and 2 (1·3 per cent.) had died of non-psychiatric illness; in the modified group, 26 (47·3 per cent.) remained well, 20 (36·4 per cent.) had minor symptoms, 7 (12·7 per cent.) relapsed and 2 (3·6 per cent.) committed suicide. Summarizing these results, a higher proportion of patients remained symptom free in the unmodified treatment group than in the modified treatment

group, a smaller proportion suffered from minor symptoms, the proportion of relapses, as measured by the need for treatment in the first year, was lower, though the proportion of recurrences was the same—assessed by considering the figures for further treatment at the eighteen month and two year periods. Less patients committed suicide, only three in the unmodified group as compared with five in the modified group over the whole period (Table VIII).

TABLE VIII
Follow-Up after One Year

Diagnosis	Unmodified E.C.T.				Modified E.C.T.			
	Symptom Free	Minor Symptoms	Relapse	Dead	Symptom Free	Minor Symptoms	Relapse	Dead
Manic-depressive psychosis	55 56.7%	33 34.1%	9 9.3%	0 0%	9 28.2%	14 43.8%	8 25.0%	1* 3.1%*
Involucional depression	15 31.9%	25 53.2%	5 10.6%	2 4.3%	15 62.5%	7 29.2%	1 4.2%	1 4.2%
Neurotic depression	43 42.6%	44 43.6%	10 9.9%	1+3* 1.0%+3.0%*	16 39.0%	20 49.0%	4 9.8%	1 2.5%
Puerperal depression	4 57.1%	2 28.6%	1 14.3%	0 0%	3 42.9%	4 57.1%	0 0%	0 0%
Total	117 46.4%	104 41.3%	25 9.9%	3+3* 2.0%+2.0%*	43 41.4%	45 43.3%	13 12.5%	2+1* 1.9%+0.9%*

Numbers * indicate deaths from non-psychiatric causes.

If each diagnostic group is considered separately the results are similar and are summarized both in numbers of patients and percentages of the total number receiving the particular form of treatment in Tables VIII, IX and X. The exception to the general pattern is in the case of patients suffering from involucional depression. In this group more patients receiving modified electroplexy remained symptom free, there are less with minor symptoms and less relapses. This may be explained either by the fact that there are relatively few cases in this group or it may be that the factor which is responsible for the differing results between unmodified and modified electroplexy does not operate in this instance (Tables IX and X).

TABLE IX
Follow-Up after Eighteen Months

Diagnosis	Unmodified E.C.T.				Modified E.C.T.			
	Symptom Free	Minor Symptoms	Relapse	Dead	Symptom Free	Minor Symptoms	Relapse	Dead
Manic-depressive psychosis	44 57.2%	23 29.9%	10 13.0%	0 0%	8 36.4%	11 50.0%	2 9.1%	1 4.6%
Involucional depression	18 48.7%	14 37.8%	5 13.5%	0 0%	12 66.7%	5 27.8%	1 5.6%	0 0%
Neurotic depression	40 51.3%	35 44.9%	3 3.9%	0 0%	13 44.9%	13 44.9%	3 10.4%	0 0%
Puerperal depression	4 57.1%	2 28.6%	1 14.3%	0 0%	3 50.0%	3 50.0%	0 0%	0 0%
Total	106 53.3%	74 37.2%	19 9.6%	0 0%	36 48.0%	32 42.7%	6 8.0%	1 1.3%

TABLE X
Follow-Up after Two Years

Diagnosis	Unmodified E.C.T.				Modified E.C.T.			
	Symptom Free	Minor Symptoms	Relapse	Dead	Symptom Free	Minor Symptoms	Relapse	Dead
Manic-depressive psychosis	37 61.7%	16 26.7%	6 10.0%	1 1.7%*	5 31.3%	7 43.8%	4 25.0%	0 0%
Involutional depression	12 50.0%	10 41.7%	2 8.3%	0 0%	8 57.2%	5 35.7%	1 7.2%	0 0%
Neurotic depression	32 53.3%	24 40.0%	3 5.0%	1 1.7%*	10 48.0%	7 33.3%	2 9.5%	2 9.5%
Puerperal depression	5 71.4%	1 14.3%	1 14.3%	0 0%	3 75.0%	1 25.0%	0 0%	0 0%
Total	86 57.0%	51 33.8%	12 8.0%	2 1.3%*	26 47.3%	20 36.4%	7 12.7%	2 3.6%

Numbers * indicate deaths from non-psychiatric causes.

Patients were also asked whether they would consider having electrical treatment again if there was considered to be a need for it. Of those who had received unmodified treatment, 25 (9.9 per cent.) would not have it again, while of those who received modified treatment, 16 (15.4 per cent.) would refuse it again. Reasons for refusing varied from being frightened of electricity to feeling that it had been of no benefit, and there were several complaints about permanent or long-standing subjective memory impairment. It was not considered possible to differentiate between those who were refusing to have further treatment on grounds of failure to be cured and those who were afraid of the treatment though accepting that they had been helped.

DISCUSSION

Introduction of the short-acting relaxant drugs was welcomed as a safe, simple method of modifying the convulsions of electroplexy, and this was considered to produce only beneficial effects. It reduced the incidence of fractures and enabled the treatment to be given to physically frail or ill patients. It was only after it had been in regular use for a considerable period in this hospital that doubts arose about the results. Some patients seemed not to respond to modified electroplexy but did so to a later course of unmodified treatment, and because of this growing suspicion this survey was undertaken with the results indicated.

These figures show a tendency for patients who receive modified treatment to make a less satisfactory recovery, after a longer stay in hospital, which may be due to a greater number of treatments required, and to relapse more often. Considering first the examination of the records of the patients' stay in hospital, it is seen that the average stay is longer whether considered for the patients as a whole or if each diagnostic group is considered separately. Similarly the average number of treatments is greater. When the patients are discharged from hospital, a higher proportion of patients receiving the unmodified treatment are discharged "Recovered" than those receiving modified treatment; the reverse is true for the category "Relieved". I wish to emphasize that I am comparing the two forms of E.C.T. rather than assessing the results of E.C.T. for different diagnostic groups, with which I am not directly concerned in this survey. I am assuming that as far as each diagnostic group is concerned, the patients receiving modified or unmodified treatment are essentially similar, and any differences will be equally represented in these two groups of patients.

In trying to explain these results two likely possibilities must be considered.

There may be some patients who are peculiarly resistant to modified electroplexy, and these results produce the artificial appearance of an overall poor effect, or else there is some feature of modified E.C.T. which produces somewhat poorer results in all patients. The evidence available in this paper is insufficient to reach a final opinion on this point but future lines of enquiry are indicated.

Examination of the four diagnostic groups revealed no essential difference in response to the two types of treatment, and a similar examination of the results, this time according to age rather than diagnosis, did not reveal a definite difference.

There was some evidence suggesting that the older the patient, the more similar were the results of the two treatments (see Table V).

These results were subjected to a three factor analysis of variance to determine the relative effects of age, number of treatments, and the type of treatment on the length of stay in hospital. Unfortunately, there proved to be such a large interaction between the three variables that it was not possible to complete the analysis. Inspection of the results suggested a suitable two factor analysis, which was carried out on those patients who had received more than average number of treatments and less than average number of treatments. In the former group, neither age nor type of treatment proved to have a significant effect in determining the different lengths of stay in hospital, but where less than average number of treatments were given the type of treatment was found to affect significantly the length of stay in hospital, modified E.C.T. resulting in the longer stay.

An explanation of this finding is that division of both modified and unmodified treatments groups into those receiving more than average and less than average numbers of E.C.T. is, in fact, also dividing them into straightforward cases and complicated or atypical cases. The average number of treatments given was 6.8. It is usually accepted that the standard course of E.C.T. to cure a straightforward depression is six and this would suggest that the group requiring less than 6.8 E.C.T. would contain most of the uncomplicated cases while the atypical cases would be more likely in the other group. It is therefore noteworthy that it is in the more straightforward group that the difference between unmodified and modified treatment is found significant, at the 0.05 level in respect to age, and when a similar analysis is carried out in respect to diagnosis it is significant at the 0.2 level.

Inspection of the records of patients who had been in hospital for a longer than average period, or received a greater than average number of treatments did not reveal any suggestion that they formed a homogeneous group. It is felt that the most likely explanation of these findings is that modified E.C.T. has less therapeutic effect, although this may only become apparent in a limited number of patients.

When patients were followed up after one year, the results of the unmodified group were better than the modified group when taken as a whole, and also when each diagnostic group was considered separately, with the exception of those patients suffering from involutional depression. In this group those receiving modified E.C.T. fared better than the unmodified group. This may be accepted as a valid finding, which cannot be explained and to which further enquiry must be devoted, or it may be due to the rather small numbers of patients in this group. A chi-square test showed the results could be explained purely on the basis of chance. However, further consideration will be given to the statistical examination of these results.

Similar findings were obtained from eighteen month and two year follow-up

though the difference between the two groups gradually became less as might be expected, as the E.C.T. effect was lost and the follow-up represented more the prognosis of recovered cases of depression.

Before one can consider why there should be a difference in the results of these two forms of E.C.T. it is necessary to know the mode of action and site of action of the treatment. Many theories, both psychogenic and physiological, have been put forward, and Hill (1954) discusses the relationship between them. He points out that clinical research does not support the view that fear, punishment and assault play a major part in the therapeutic effect of the treatment. In this series fear of the treatment as expressed by refusal of treatment when offered, or subsequently, when the patient was asked if she would be prepared to have it again if it were thought necessary was approximately equal in the two groups, with a rather higher percentage of the modified group refusing than the unmodified group. Thus, modifying the treatment does not seem to remove the anxiety associated with it, nor does there seem evidence that it is this anxiety which is responsible for the difference between the two forms of treatment.

Roth (1952) has suggested the biological efficacy of the epileptic fit as an apparatus for the maintenance of a relatively stable pattern of activities in the brain, and that E.C.T. is a therapeutic reproduction of this action. The action, he suggests, takes place in the upper mid-brain, and he has pointed out that it is in this region that barbiturates have an action. Barbiturates are well-known anticonvulsant drugs and I feel that they may "protect" this region from the therapeutic effects of E.C.T. The epileptic fit certainly takes place when anaesthetic and relaxant are used as shown by Piekenbrock *et al.* (1956) but there may well be something lacking. A recent paper by French workers (Morin and Corriol, 1957) shows one of the effects which are lacking though not suggesting that this is responsible for the lack of therapeutic effect. They had found in animals that a well-marked hypertension develops dependent on the "pressor area of the basal diencephalon" which has to participate in the seizure. This diencephalic activity is inhibited by small doses of anaesthetics or chlorpromazine which do not themselves modify the cortical seizure. The peripheral mechanism of the hypertension was found to be dependent upon neurogenic vasoconstriction which may be accompanied by liberation of adrenaline.

If it is correct that it is the anaesthetic which is at fault, it is logical that treatment should be given without an anaesthetic, but with a relaxant. This has of course been done (Gillie and McNeill, 1955; Impastato, 1956) though results of this treatment have not been compared with those of other methods. However, an ethical point is raised by the use of this method. Vivid accounts have been given of how a volunteer feels when he receives a dose of relaxant without anaesthetic; a feeling of suffocation and of being quite unable to inform those around him of how he feels. Presumably a patient experiences the same discomfort when he is given it prior to E.C.T. but because of the retrograde amnesia produced, does not recall it. Does it do any harm, either by increasing the likelihood of refusals, or by some long-term traumatic effect on the psyche, to undergo a series of painful experiences, even if they are not remembered afterwards? In any case is it ethical to give such treatment even if one knows the patient will not remember it? This difficult question is one which must be left for the individual practitioner to answer. My own feeling is that if the anaesthetic is at fault, another must be found which does not have this "protective" effect. Perhaps the new steroid anaesthetics will provide a solution to this problem.

If it is the relaxant which is at fault, this suggests that either the relaxant has some undescribed central effect, or that the actual peripheral manifestations of the fit are responsible for its therapeutic effect; in other words, release of lactic acid or other metabolic products into the blood stream. This theory was proposed by Lowenbach and Greenhill (1947) but has not received general acceptance.

The question must be asked whether it is important that a patient receiving modified E.C.T. should spend a few days longer in hospital. To do this one has to weigh up the advantages and disadvantages of the two types of treatment. Unmodified E.C.T. undoubtedly produces a higher incidence of fractures, but is otherwise relatively safe. It is quicker to administer and fewer drugs are necessary, so that less complications are likely. Modified treatment requires a more experienced administrator, some would say an anaesthetist, and there are more drugs given, with dangers of intra-arterial injection, prolonged apnoea, and laryngeal spasm. It is somewhat more time consuming, but these difficulties are not serious, and if results are equally good, I think that modified treatment is to be preferred. However I have indicated that the results are not the same, though in this paper I am unable to say why they should be different. If it is merely a question of a few more days in hospital, one or two more treatments, it would not be important: but I feel these results reflect the fact that some patients respond poorly, if at all, to modified E.C.T., and I do not know how these patients can be recognized, except by failure to respond to treatment. Some patients may be discharged from hospital less well than they should be, and it would appear that a proportion commit suicide. I feel, therefore, that this is not a matter which can be lightly passed by but one on which further investigation is needed.

This enquiry is not considered complete in itself, but rather a pilot study suggesting the need for further investigation by controlled experiment. The method of retrospective examination of records and results has many fallacies, while some of the assumptions made may not be completely valid. Thus, there is the possibility that one doctor would treat all his patients by a long course of modified E.C.T. and keep them in hospital for a long period. Or it may be that only cases with poor prognosis were given modified E.C.T.—perhaps because of concomitant physical disease. As explained earlier, this is probably not so; working at this hospital during the period under observation, I feel that the doctors tended to deal with similar patients in similar ways.

The results given in this paper had applied to them tests of significance where shown. In many cases these results were shown not to reach a satisfactory level of significance. This may be thought to invalidate the findings of the paper except for the fact that all the results show a tendency in the same direction, with the exception of the follow-up results in involutional depression. This uniform tendency of a group of results which are to a large extent independent, makes the conclusions drawn from them more likely to be valid, though requiring further confirmation.

I have shown that patients tend to respond less well to modified E.C.T. although I have not been able to prove this. I have suggested that the anaesthetic is the most likely drug which lessens the therapeutic effect, although the result may be dependent on some factor in the patient, which may make him more susceptible to its action. Finally, further enquiry by controlled trial is desirable to prove this point and also to throw light on the mechanism of action of what is still almost entirely an empirical treatment.

SUMMARY

1. An examination is made of the results of unmodified and modified E.C.T.
2. This was carried out by retrospective examination of hospital records after discharge and a follow-up at least of one year of all female patients suffering from depression and treated by E.C.T.
3. The results showed that patients who were treated by unmodified E.C.T. were in hospital for a shorter period, received less treatments and were more likely to remain well than those treated by modified E.C.T.
4. The results are discussed and thought to indicate the need for a controlled trial of unmodified and modified E.C.T.

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UNILATERAL ELECTRO-CONVULSIVE THERAPY*

By

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PREVIOUS WORK

Introduction

ELECTRO-CONVULSIVE therapy was first introduced by Cerletti and Bini in 1938, and has now become the principal physical method of therapy in psychiatry.

Unhappily, whilst this method of treatment is at present enjoying such deserved popularity, it is not without undesirable effects, some of which may be quite insidious.

Modification by relaxants has resulted in a change in importance of its complications and now in a well-organized unit the old complications of dislocation of bones, crush fracture of vertebrae, broken teeth, aspiration pneumonia, and lung abscess, are rarely encountered. There are, however, other complications, some theoretical, though with possibilities that cannot be ignored, and others of major practical importance.

Cerebral Impairment

Of practical importance is the finding that when an electric current is passed through the brain in the conventional manner, severe mental impairment occurs.

Pacella *et al.* (1942), showed that the electroencephalograph develops abnormalities consisting of 3-6 cycle/sec. slow activity of moderate to high voltage, which may persist for several weeks, depending on the amount of treatment given.

Juba (1948), described Gerstmann syndrome, consisting of finger agnosia, acalculia, agraphia, and disturbances of right and left laterality, being often encountered for a short period after the shock.

Stengel (1951), draws a parallelism between repeated shocks and head injury, and during his condemnation of intensive electro-shock therapy as a method of treatment, states that amnesia can be gross, extending over several years.

* Based on a paper given at the Quarterly Meeting of the Royal Medico-Psychological Association at Bristol in May, 1957.

Anderson (1951), states that electro-convulsive therapy should be avoided if possible in very superior intellectuals. He cites examples of poets who ceased creative work following electro-convulsive therapy, and professors and students who forgot large parts of their knowledge.

Hafner (1951), believes that after eight or even less shocks, early organic syndromes may commence. These manifest themselves as apathy and confusion. He states that to prevent amnesia must be a major objective of therapy.

These memory disturbances may have a legal significance in that patients may forget for a time that they "want to go home" and therefore the treatment may constitute a means of restraint, whilst the disorientation and confusion increase significantly the observation needed of patients given treatment on an out-patient basis.

Stengel (1951), showed psychoneurotics and Taylor and Pacella (1948), showed arteriosclerotics particularly susceptible to memory disturbances after electro-convulsive therapy.

One theoretical complication which is little known, and we feel must be stressed, is that of eye lesions.

Eye Changes

Duke Elder (1954), states that lenticular opacities have occurred following exposure to high voltage alternating current, direct current, and electro-shock therapy. He stresses the following points:

1. After injuries due to lightning, opacities develop in 1 to 7 days. After strong industrial currents an average of 2-4 months with extremes of days to years—cases after 3, 6, and 11 years are reported.
2. The strength of current has varied from 500 to 50,000 volts, though the development of cataract seems to bear no definite relationship to the strength of current.
3. If bilateral cataract follows one-sided contact points, then changes in the proximal eye precede those in the contralateral eye by several months.
4. Other eye disturbances associated with the passage of electric current include skin burns and necrosis—painless and aseptic, hair singeing, spasm of accommodation, photophobia and blepharospasm, contraction of visual fields, and retinal and corneal lesions.

Though we personally have never witnessed any serious eye lesions following electric shock therapy, photophobia, accommodation difficulties, and skin burns have frequently been noted. The long latent period of cataract means that it will be many years before a verdict of innocence can be given.

It was with the object of reducing the confusion and memory disturbances and preventing the passage of the current through the orbits, that the unilateral application of E.C.T. was started by us.

Induced Focal Fits

There are some previous references in the literature to induced focal fits—Kalinowsky (1952), states that if focal fits occur during the application of conventional E.C.T. they are not as therapeutically effective, but there is a much quicker return to consciousness than when a generalized convulsion occurs.

Pacella and Impastato (1954), used unilateral application of the electric

current but used continuous current gradually increasing until the contralateral seizure commenced. They stimulated first one side of the brain and then the other. They thought focal seizures were less therapeutically effective than generalized, but were useful in poor risk patients, the elderly and paranoid cases who need less confusion of consciousness.

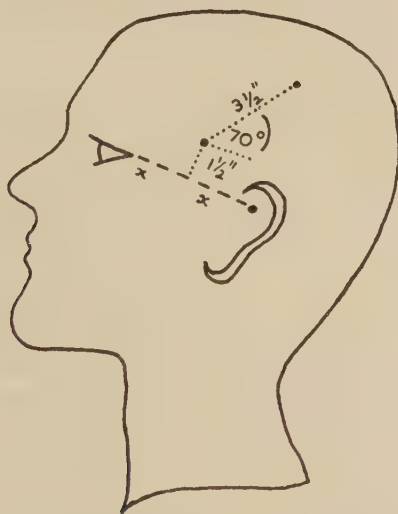
EXPERIMENTAL TECHNIQUE

1. METHOD OF APPLICATION

All patients were premedicated with oral atropine 1/75th about 45 minutes previous to intravenous pentothal 150 mg. and Brevdil E 50 mg.

The electroplexy machine used had electrodes about $1\frac{1}{4}$ in. diameter and was of the type where discharge of a condenser gave a constant amount of current to each patient.

The electrodes were lubricated with Cambridge electrode jelly as also was the scalp at the points of application. The non-dominant side of the skull was chosen since it was felt that damage here would not be as important as on the dominant side. The lower electrode was midway between the lateral angle of the orbit and the external auditory meatus and $1\frac{1}{2}$ in. above this line. The upper electrode was 3 in. higher than the lower and at an angle of 70° to the line. (Slight deviations from these points do not materially affect the result.)



After the passage of the current, an air way was inserted. Usually respiration commenced again within 90 seconds, but if this was not so, positive pressure insufflation by means of a Beaver respirator was given.

2. (a) ASSESSMENT OF UNDESIRABLE EFFECTS

This involved the use of three assessors, each provided with a clock or watch zeroed just previous to commencing each day's procedure.

1st Assessor

Showed the patient a card on which was written a short memory test sentence containing four items. He read the sentence to the patient and got him to repeat it. He then recorded:

Name of Patient.

Time of Injection of Pentothal and Relaxant.

Time of applying the electric shock.

Details of method of application, i.e. Unilateral or Bilateral.

Result, i.e. Subshock, Focal Fit, or Generalized Fit.

2nd Assessor

Recorded:

Name of Patient.

Time of first attempting to swallow.

Time of first attempting to breathe.

3rd Assessor

Recorded:

Name of Patient.

Orientation in Ego, i.e. Time of knowing Name and Age.

Orientation in Place, i.e. Time of knowing where he was.

Orientation in Time, i.e. Time of knowing Day and Date.

Memory test, i.e. If he could remember the sentence he was shown just prior to the treatment.

Due to practical considerations, the maximum time for observation was 15 minutes in each case and if a particular faculty had not returned in that time it was recorded as 15 minutes+.

2. (b) ASSESSMENT OF DESIRABLE EFFECTS

1. Patients for whom E.C.T. was recommended were carefully assessed prior to commencing therapy and a rating on a 4 scale grade (0=Nil, 1=Slight, 2=Marked, 3=Extreme) given for each of the following symptoms:

Stupor	Misery
Anorexia	Feelings of Unreality
Insomnia	Retardation
Weeping	Hallucinations
Ideas of guilt, worthlessness, and general worries.	

The score of the individual items for each particular patient was then summated and this total became "the depressive quotient" for that particular patient.

2. Particular care was taken that the assessing doctor was not aware whether unilateral or bilaterally induced E.C.T. was given. This was done by means of a draw system and a series of cards in which the nursing staff played a prominent role.

3. A total of four E.C.T.s were then given and the depressive quotient again determined in a similar manner as previously.

RESULTS

Undesirable Effects

A total of 135 electric shocks were analysed. These consisted of:

39 cases of Bilateral E.C.T.

31 cases of Unilateral E.C.T. with generalized fit.

33 cases of Unilateral E.C.T. with focal fit.

32 cases of Unilateral subshocks.

TABLE A

Type of Fit	No. of Cases	Preceding Passage of Current	Arithmetic Mean in Minutes						Memory Recall Test
			Following Passage of Current						
			Injection	Commenced to Swallow	Commenced to Breathe	Orientated for			
						Name	Place	Time	
Bilateral induced fit	39	0.62	0.82	1.08 s=0.57	9.83 s=4.28	13.86 s=2.38	14.47 s=1.26	1	
Unilateral induced general fit	31	0.64	1.18	1.28 s=0.76	6.07 s=3.59	8.07 s=5.26	8.68 s=5.2	19	
Unilateral induced focal fit	33	0.67	1.07	1.59 s=0.87	4.36 s=3.38	5.05 s=4.36	6.17 s=4.5	27	
Unilateral induced subshock	32	0.63	1.06	1.6 s=1.00	3.0 s=1.78	3.0 s=1.97	4.41 s=4.16	24	

Whilst certain of the above clinical impressions could not be confirmed statistically (because of high values of standard deviation and small differences in arithmetic mean necessitating larger number of cases), the following results were noted:

1. The breathing commences quicker after a generalized fit induced either bilaterally or unilaterally than after a unilateral subshock or focal fit. (Strong clinical impression.) Probably due to greater anoxia and accumulation of carbon dioxide after vigorous muscle jerks.

2. Orientation for name and age returned quicker after a unilaterally induced generalized fit than after a bilaterally induced one. (Highly statistically significant.)

3. Orientation for time and place returned quicker after a unilaterally induced focal fit than after a unilaterally induced generalized fit. (Statistically significant.)

4. There was a definite order for return of orientation in all spheres. (Strong clinical impression.)

Subshock earliest.

Unilaterally induced focal next.

Unilaterally induced generalized.

Bilateral induced generalized last.

5. Recall of the test item was much higher in all the unilaterally induced cases than in the bilaterally induced ones. (Highly statistically significant.)

6. After a bilaterally induced convulsion features of cerebral concussion, such as automatic behaviour, dazed expression, and restlessness were more prominent than after a unilaterally induced one. (Clinical impression.)

Desirable Effects

A total of 43 patients were studied and each given a course of four electric shocks.

They consisted of 15 patients given bilaterally induced generalized fits, 21 patients given unilaterally induced generalized or focal fits, and 7 patients given unilaterally induced subshocks.

On analysis these results show:

1. No statistical significant difference between the improvement caused by bilaterally induced generalized fits and unilaterally induced fits. (There was however, a clinical impression of slightly better improvement and more complete remission with the bilaterally induced fits.)

TABLE B

Type of Fit	No. of Cases	Arithmetic Mean of Depressive Quotient		
		Before Treatment	After Treatment	Improvement
Bilateral generalized	15	7.8 (s=3.8)	2.3 (s=2.5)	5.5 (s=3.9)
Unilateral generalized or focal fits	21	8.4 (s=3.5)	3.9 (s=3.3)	4.5 (s=2.8)
Unilateral subshocks	7	9.1 (s=3.2)	7.1 (s=5.5)	2.0 (s=2.8)

2. Cases treated with Sub-Convulsive Therapy showed a statistically significant less improvement than those treated with bilaterally or unilaterally induced generalized or focal fits. (Statistically significant.)

DISCUSSION

The above are the facts and one has now to consider what deduction can be drawn from them.

The indication for unilateral E.C.T. is when one needs minimal side effects with not maximum therapeutic response at first. It would seem that a large percentage of cases would fit into the category and include the following:

1. Elderly patients with severe arteriosclerosis who are depressed and difficult with food.

2. Patients of very superior intelligence, and especially those who have to earn their livelihood with retained knowledge.

3. Young patients under the age of 40 years who show predominant neurotic features and yet who are depressed sufficiently to warrant E.C.T.

Patients to whom we would give bilateral E.C.T. are:

1. Involutional depressives of average intellect.

2. Severe depressives who are actively suicidal (on Suicide Caution Cards).

3. Any patient who after six unilateral treatments is still being difficult with food and showing other features of depression.

4. Catatonic Schizophrenics who are dangerously impulsive.

Future Trends

Two particular lines on which further research would appear to be fruitful are:

(a) Close studies of patients having had E.C.T. several years before for eye lesions. This point should be definitely cleared up one way or the other as soon as possible.

(b) Finding of a slightly larger number of Unilateral E.C.T.s has the same effect as slightly less number of Bilateral treatments.

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Reviews

Analysis of Perception. By J. R. SMYTHIES, M.A., M.D., M.Sc., D.P.M. Routledge and Kegan Paul, London, 1956. Pp. 140. Price 21s.

The study of perception, and its relation to the very diverse fields of philosophy, epistemology and neurophysiology, is a most difficult one. Few who have studied the subject have brought to it an adequately wide experience. Dr. Smythies, however, is a psychiatrist who is at present conducting research in physiological psychology, has worked in neurophysiology and anatomy, and has studied philosophy at some length. He has also worked with hallucinogens, and was one of the team responsible for the discovery that trimethoxyamphetamine was in this group. He is therefore unusually well qualified to write on the subject.

Such a subject, treated with the seriousness that it deserves, cannot make easy reading, and this book is no exception. It bears throughout, however, the mark of hard and prolonged thinking. It is clearly written, and should be studied by those who are interested in the subject.

W. ROSS ASHBY.

Probability and Scientific Inference. By G. SPENCER BROWN. Longmans, Green & Co., London, 1957. Pp. 154. Price 15s.

The author, a professional mathematician, believes that something is rotten in the state of Statistics, and in this book he attempts to display various inconsistencies, paradoxes, and other confusions that he believes to infest the subject. Plenty of confusion is certainly displayed in the book, though whether it is due to the subject or to the author's failure to think slowly and carefully must be left to the reader to judge.

The subjects of statistics and probability are not easy, and quite elementary questions are apt to provide tangles that need much thought for their straightening out. Those who like puzzling themselves will like this book, but the reviewer will pay closer attention when Mr. Brown stops stirring up difficulties and proposes some solutions.

W. ROSS ASHBY.

L'Homme Criminel. By DR. ÉTIENNE DE GREEF. Nauwelaerts, Louvain, and Beatrice—Nauwelaerts, Paris, 1956.

This is a symposium in honour of Étienne de Greef, a Belgian Criminologist (and clinical psychiatrist) of great repute—much greater abroad than at home we are told by Professor Leclerc, who opens the papers with a glowing critique of the man to whom the volume is dedicated, and who, much later on, contributes a major paper on the immediate future of criminology.

The book is divided into three sections: (i) the personality of the delinquent, (ii) psychiatry in the penitentiary, (iii) psychiatry and Social Defence (the contriving of a healthy social system, including the retrieval of all criminals).

The contributors are mainly pupils of Professor de Greef, at any rate in the first section, and they both readily acknowledge their indebtedness, and enthusiastically delineate the impact of their master on Belgian and on all French-speaking criminological thought. If to a non-Gallic reviewer the tributes are, at times, effusive, and seem to impede the otherwise very free thought, their sincerity is patent, as is the pleasure with which they are made.

The section devoted to the personality of the criminal contains five papers; the last being a tripartite discussion on responsibility, by a jurist, an anthropologist and a psychiatrist. On the whole, the very general, theoretical, philosophically flavoured and somewhat doctrinaire quality of this section is much relieved by Professor

O. Kinberg's contribution on the "Biological infrastructure of the criminal act as a basis of an objective criminogeny". He fails, however, to carry conviction that an expansion of Lhermitte's observations on punctate cerebral damage in encephalitides, married to an hypothesis of afferent and/or efferent pathway interruption can yet, or in the visible future, lead us to the understanding implicit in the title—the more so, as Professor Kinberg steadfastly asserts that in the neuro-cerebral pathways there is no point at which the psychic circuits can be intercalated. But let it be said that Dr. Kinberg puts forward his ideas as foreshadowings and no more.

The second section, the psychiatrist in the penitentiary, opens with a racy attack by the First Secretary to the Ministry of Justice—M. Cornil, upon the practical contribution actually made by psychiatry and psychiatrists to the day-to-day problems confronting the prison administrators in dealing with prisoners. It is much more stimulating reading than the reply which comes later. Much of this section has to do with Belgian domestic matters, but Miss Tuerlinckx, a social worker, the first female to work in the Central Prison at Louvain, the site of Dr. de Greef's life-long labours, is given the opportunity, and takes it, of recalling her first reactions to her work which she succeeded in being allowed to undertake. There is also included a chapter on case history taking and Extended Observation on children.

It is in this section too, that Dr. E. de Greef writes his paper, essentially a consideration of three men, each of whom murdered his wife. The material is of great interest, and its interpretation by Dr. de Greef, open to much argument. He notes, for instance, that in two cases the homicidal act was quite out of character with the general life pattern of the culprit, and that the prisoner was admitted in a state of mind and outlook which was foreign to him, slowly resolved in two or three years or more and could then be seen to have existed for a significant time before the crime, insidiously worsening. Dr. de Greef sees all this as evidence of a moral deterioration in the individual, which is capable of recovery if he be put in satisfactory conditions; prison (and, according to the criticism of M. Cornil, solitary confinement), being satisfactory. Dr. de Greef indicates that his contacts with these three chosen cases are few and far between.

The last section of the book is devoted to medico-legal considerations by psychiatrists, including contributions by Zilboorg, who touches on some of the main points made in his Isaac Ray memorial lectures (reviewed in these columns about 15 months ago) concerning the evolution of anglo-saxon medico-legal thought on the topic of legal tests of responsibility. He brings matters up to date by quoting the 1954 reformulation of an American Court—that of Columbia—in which the establishment of mental illness and the origin therein of the criminal act, becomes the test to be applied.

Much hard thinking and writing has gone into this tribute to Professor de Greef, and there is much that is admirable, but some of it is obscured by the generality of approach and argument, and by the lack of clarity of the topic of argument, when murder, delinquency and criminality are used without indication with separate and synonymous meanings.

JONATHAN GOULD.

Alcoholism. By LINCOLN WILLIAMS. Livingstone, Edinburgh and London, 1956.

Dr. Lincoln Williams, in an essay of some 25,000 words, has attempted gallantly, and with considerable success, to convey an impression of that field of concern that is called alcoholism. At a time when psychiatric literature is not specially notable for its freshness and spontaneity, it is pleasing that Dr. Williams' monograph retains much of the flavour of cursive discussion, such as might go on between more and less experienced physicians engaged in the same field. It is this quality of directness, of lack of dogmatic authoritativeness, carrying yet that air of confidence of one who knows from a wealth of experience, which leads the reader to forgive with ease the occasional digression of the short chapters, from the title, to other no less interesting topics. And the chapters are short—at times, less than 500 words! Indeed, the book,

which is well printed and easily read in an afternoon, is described as a manual—and could well serve as a light comment on some trends in medical literature nowadays.

The author, like all workers in such a field, has his special interests, and these persuade him to give a relatively extensive consideration to certain methods of treatment (which are admittedly successful only in the hands of the much experienced) and yet to give insufficient information for the tyro to undertake the techniques. Nevertheless, the general approach is stimulating and interesting—and is redolent of clinical practice, whence, once, the best medical literature sprang.

JONATHAN GOULD.

Etiology of Chronic Alcoholism. Edited by OSCAR DIETHELM. Published by Charles C. Thomas, Springfield, Illinois and Blackwells, Oxford, 1955.

A symposium on the *Etiology of Chronic Alcoholism*, edited by Dr. Oscar Diethelm, who also planned and directed the research programme, of which this book is the first-fruits, after some six years of labour by a large team representing several disciplines, approaches the problem from various aspects. These include clinical psychiatric descriptive studies, genetic studies including a comparison between American and Swiss patients; a social anthropological study of a quasi-segregated Chinese community in New York, in which alcoholism was not a significant problem, and a chapter on the biochemical characteristics of the blood of the alcoholic—giving perhaps new significance, in due course, to the phrase “the hair of the dog . . .”. The whole, is introduced by a chapter by Dr. Diethelm, in which he explains the premises of the work, defines his terms—particularly “chronic alcoholic” and “excessive drinker”—which definitions are employed in each succeeding chapter, being re-stated by the contributor.

Dr. Diethelm draws the broad picture of the work, indicating that there were aspects, such as the Rorschach studies, which could not be prepared in time for publication, but which he draws on, to the reader's interest, as in commenting that only four of the patients studied (76 current, and 85 case histories) corresponded to Rorschach's original description. The comparison between Rorschach results and biochemical investigation, however, each carried on independently of the clinical psychiatric study and of each other, gives positive agreement in a surprisingly high proportion of instances, where emotions of anxiety or resentment were concerned (90 per cent.). If Dr. Diethelm has a special interest in any aspect of his symposium, it would seem to be in connection with the work, reported by M. Freile Fleetwood, in the chapter on Biochemical Experimental Investigations of Emotions and Chronic Alcoholism, in which there is adduced evidence in support of the claim that there are to be found distinct chemical substances in the blood, in association with, and quantitatively varying with the intensity of, the moods of anxiety, tension and resentment.

These substances are (nor-) adrenaline-like, acetyl-choline-like, and another, dubbed “resentment-substance” which has an acetyl-choline-like effect on certain animal tissue preparations only after these have been submitted to the action of hyoscyamus with consequent destruction of acetyl-choline effect. (An acetyl-choline like compound is considered the tension substance.) The material offered in support of this thesis, is immense, and perhaps not readily assimilable to the clinician, who might well feel that, at times, generalizations are made from single instances, and that the whole thesis of “resentment-substance”, to which alcohol is deemed a specific biochemical and psychic antidote, is founded upon the responses of nine out of ten alcoholic patients, to whom whisky (six ounces) was given, four hours after breakfast. Another alcoholic patient became more resentful and suspicious, as was understandable in view of her history, and the concentration of “resentment-substance” rose. In two other alcoholics, who were atypical, in that they were not resentful, but anxious people, the concentration of anxiety-substance, not “resentment-substance”, was mainly affected. The general flavour of this chapter is that the biochemistry accounts for the psychic state, and that the alcoholic is distinctive in that he generates

"resentment-substance"—to which alcohol is a specific antidote. Several cases which would suggest the opposite, that the resentment-substance found by Fleetwood was produced as a somatic response to the psychic state, are mentioned, and then disregarded. The correlation between biochemical findings and psychiatric findings appears to be 100 per cent. with regard to all three substances, and this elicits no comment from the author, and although he notes that some people are unaware of their emotions, he seems to overlook both repression and disassociation as sources of biochemico-psychic discrepancy. Neither does he draw any inference from the discrepancy noted in a technician, who, having been found to have "resentment-substance" in her blood when apparently urbane, admitted to covert resentment regarding the promotion of her colleague. Finally, the effect of six ounces of whisky was to banish "resentment-substance" and resentment, for more than 24 hours, except in two cases, where up to ten ounces proved necessary. This observation serves to throw enough light on the natural history of chronic alcoholism, to show how far from understanding are we—in a biochemical sense—the drinking bout which goes on for days and involves more than ten ounces of spirit! Clearly, more problems are raised than solved by Dr. Fleetwood, but this is research, and it is to be hoped that in the further edition of this book, to which one would look forward in due course, the few blemishes in the format of this chapter (such as the presence both of a diagram and a photograph of the experimental apparatus employed, to neither of which is there appended a description of the components, the function of some of which are not at once clear to the Clinician; and the omission of the designation of Figure 2) may be removed from an otherwise stimulating contribution.

The clinical psychiatric studies of 161 cases (76 current and 85 derived from over 400 records), presented by Dr. Sherfey, have been based upon material investigated with particular assiduity. The criteria of acceptability of the case histories of past patients were stringent, as is indicated by the acceptance of less than 1 in 5 case records of alcoholics as adequate in detail and extent. On the basis of this material, Dr. Sherfey divides the psychiatric hinterland of alcoholism into eleven syndromes, five of which are orthodox psychiatric diagnoses, which, however, account for rather less than half the number of patients. The larger half is divided into syndromes, most of which are sex-linked, and are combinations of terms indicative of emotional maladjustment.

Dr. Sherfey's conclusion is that alcoholism is a symptom or mode of reaction to stress with no characteristic or specific psychiatric prerequisites, although an adequate recognition of the psychiatric concomitants is essential in the evaluation of the given case. She also makes a point, which is, perhaps, not given adequate prominence (although it fits in very well with the implications and conclusions of the anthropological survey), that alcoholism in women increases with social acceptance of woman's consumption of alcohol in public.

The force of the social sanction with regard to alcohol, its use, in varying degree and circumstance, and, finally, the power of the social sanction to prevent, almost completely, the abuse of alcohol in the form of chronic alcoholism, is drawn, in interesting fashion, by Dr. Barnett, in a social anthropological study of the 5,000 or more Chinese who form a quasi-segregated community in New York. These people have retained through two generations, so far, almost intact, their original value-systems, which prescribe, clearly and benevolently, those occasions when alcohol may be consumed, and, at times, very liberally. The social recoil, or even revulsion, from one who fails to recognize his limits, and who cannot, therefore, desist while still in control, may take many forms. Women are not permitted as much access to alcohol, as are men, and a woman the worse for drink is the greater disgrace to herself, her family, her sex and her community. Interest in consumption of opium and the like was scrupulously avoided.

This is a very ably executed piece of observation and reporting, which is, admittedly, only an account of the first-hand impressions formed by the author during six months of field-work among the people of New York's Chinatown. The psychiatrist

cannot but be interested in a report such as this, and would greet further instalments with pleasure.

The clinical psychiatric *pièces de résistance* in this volume, are the contributions by M. Bleuler, who spent a year with the workers at the Payne-Whitney Clinic, where Dr. Diethelm is Director. Here, Dr. Bleuler made his own further examination of 50 American Alcoholics undergoing treatment. By virtue of the criteria for admission to this Clinic and its associated beds, the patients were of the upper intellectual and occupational strata. Some of Dr. Bleuler's conclusions must be quoted:

Schizophrenia, manic-depression, epilepsy and oligophrenia, are no more common among the siblings and antecedents of alcoholics than among the average population of the same social and economic class. But alcoholism is much more common among siblings, antecedents and spouses, and so are morbid personalities, than in the average population of the same social and economic class. Most of these findings may be generalized, in view of his own further work in Switzerland (of which he contributes a preliminary discussion in a further chapter), and, also, the work of others which he quotes to effect. He uses his studies on alcoholism to refute the postulate that homosexuality is a necessary precondition, and to make cogent argument concerning those psychodynamic theories of schizophrenia, which causally relate this condition to stress in the mother relationship. Endocrinopathy may, in the given case, be a relevant factor in alcoholism, either as a causative metabolic lesion, or as a focus of emotional disturbance. Finally, the genetics of alcoholism are the genetics of abnormal personality development, of which alcoholism is mainly to be considered a symptom. But only an indication can be given here of the clinical satisfaction to be found in reading these lucid chapters, despite their far from infrequent impressive tables of figures, which the text brings alive.

Thus, the picture emanating from these pages is the alcoholic as a person, still more commonly male, who, the carrier of emotional maladjustment, of more or less formal and overt sort, responds to the stress of living, created by his environment and his reaction to it, by a self-damaging form of behaviour familiar to him in his near-ones in his formative years, and permitted by the society in which he finds himself. "Drowning one's sorrows in drink" has become transformed into "resolving resentment in rye", and all the usual and familiar adjuvants of the anamneses of maladjusted ones are present, in profusion, in the pasts of these. To boot, there may be somatic strains—perhaps endocrine and perhaps metabolic—but the main impression of the volume is that the aetiology is to be found in social, cultural, familial domestic and psychogenic spheres, if in the latter it be permitted to include those psychic phenomena which are temperamentally determined, and all pivoting upon the generation of resentment, and "resentment-substance" to which alcohol is the antidote.

Dr. Diethelm deserves full admiration for his inspiration and leadership of this team of workers, far greater in number and diversity of discipline than the list of official contributors. He has been midwife to a volume which will interest every clinical psychiatrist, whether he have a special interest in alcoholism or not—and a volume which has sections to give much food for thought to all who are interested in the misuses of one of the greater gifts to man.

Dr. Diethelm mentions, in his opening chapter, that the work so far done has provided clues for more investigation, and Dr. M. Bleuler records the wider project on which he is now engaged at the Burgholzi. Their further reports are matters for anticipation.

JONATHAN GOULD.

The Integration of Behaviour. By T. M. FRENCH, M.D. Vols. 1 and 2. The University of Chicago Press, and Cambridge University Press, 1952, 1953.

These two volumes, to be followed by three more, set the stage for the test study—an exegesis of the day-to-day notes of a case treated for two years psycho-analytically—by considering basic postulates and the function, discerned in dreams, of the process of integration. This is an innovation in psycho-analytical thought, briefly summarized in Volume 1. "First, the motivating pressure of a need seeks

discharge in diffuse motor activity. Next, hope of satisfaction, based on present opportunity and on memories of previous success, stimulates the integrative mechanism to form a plan for realizing this hope. Finally, hope of satisfaction activates this plan so that it exerts a guiding influence, concentrating motor discharge on efforts to put the plan into execution."

Written in a slowly moving fashion, assumptions abound, each quite clearly introduced. The impression is of a labour of love for the converted, in whom alone would there be sufficient zeal to persist to the "Biological Foundations of Behaviour" at the final end.

JONATHAN GOULD.

Battle for the Mind. By WILLIAM SARGANT. Heinemann, 1957.

This delightful scientific true-life ghost story of our present day and age stole seven sunny hours with the same persuasive ease, and the same promise of a possibly happy ending, as if the tale had been told by the Maker of Dreams.

Dr. Sargent, on careful scrutiny, has nowhere, it would seem, overstepped the strict limits of scientific fact, if such a term may, even though doubtfully, be applied to clinical, essentially uncontrolled, observation. He would appear, however, to have used his language in such a way that this strict adherence is far from patent much of the time, and though, for instance, in two specific places, he explicitly states that he draws only analogy between dog-Pavlovian and Clinical, Political and Religious phenomena, yet the cursive reading of the text creates undeniably the sense that homology between the human experience and the animal response obtains.

Dr. Sargent's thesis is that the Pavlovian experimental data and theory developed in the study of dogs can, without modification, be applied with advantage to the study of various human responses, to wit:

(a) The acute battle neurosis and the response of this to abreactive methods of treatment.

(b) The phenomena of Religious Conversion—particularly in its more stormy emotional aspect.

(c) The phenomena of politically, and/or "police-fully" inspired brain-washing, confession-eliciting and thought control.

In that dogs are not credited with mind—particularly Egos, Ids, and Superegos—to draw on Dr. Sargent—a physiological explanation for the behaviour of the animals is the most apt, and in that there are similarities in the patterns of emotional response to be discerned, between the animals and the human, it is the germ of his thesis that similar physiological principles may be found to underlie the lot.

This is no statement to abash psychiatrists, doctors, most school teachers, animal trainers or dog lovers, and yet this book will arouse much controversy—in the opinion of the reviewer—because, throughout the text, the implication is, except when a formal statement of the position is made, that the observable behaviour of the dog is an adequate framework of reference for the experience of the human, neurotic, political, sinful or saved.

And it is important to remember that this book is written for the lay public. Perhaps to carry its message, and to have impact, the book had to be written in this fashion—for Dr. Sargent, while exposing, from his own diaries, Wesley, in more urbane and subtle fashion follows the precepts of this fiery reclamer of the souls of men. He provokes the reader, in part, by repeatedly directing his eye to his mirrored wagging tail, to some degree of emotionally charged attention to the topic of death, mutilation, disaster and worse—the betrayal of oneself and one's truths. He does this, whether it be in the chapters on war memories and breakdown into gibbering fear, the descriptions of conversion singly or en masse, with details of behaviour not in keeping with our cult of emotional reserve, or with accounts, mainly first-hand, of the evidence of some who have been through the brain-washing experience. Organically related to these has been a middle section on the emotional, mystical and religious states, which can be provoked by fairly simple group and sensory techniques in

voodoo and snake-handling cults. (A chapter on Brain-washing in Ancient Days is also contributed by Robert Graves, who assisted Dr. Sargant in the preparation of the final manuscript.) Having surveyed this vast territory, and having portrayed one's vulnerability to "conditioning" to "transmarginal stimulation" to "debilitation" and "ultra-paroxysmal" responses, Dr. Sargant sees to it, following Wesley, that the reader is not left in the Slough of Despond—his self-confidence and sense of self fragmented into the four aspects of canine cerebral reflex disintegration, described by Pavlov, and exploited by the Russo-Chinese terror systems and the New York Police, but leaving the role of the scientist—Dr. Sargant declares himself a lover of humanity and a saver of psyches if not of souls, and, taking us by the hand, gives good advice for the withstanding of the technique of brain-washing and the risk of falling into the pit of unwelcome political indoctrination.

This is a fascinating book, and far from flawless. Dr. Sargant does not give the evidence, so important for the layman, whereby Pavlov claimed to *show* that his dogs' behaviour was specifically localized in the cortex, and indeed, the current views of circuit-function in the brain would hardly be consistent with such ideas. Nor does Dr. Sargant explain to the layman in which way the Pavlovian theory (of stimulation—excitement—inhibition-inertia, transmarginal, paroxysmal and ultra-paroxysmal, stimulation and response, together with spreading inhibition and protective inhibition—which may become (in the human!) divorced from reality)—is in its various components more directly corroborated by neuro-physiological observation than is the equally, or more, flowery psycho-analytical theory, which makes no claims to physiological foundation in a specific sense. For Pavlov grew a great number of terms to account for the behaviour of dogs, and they but nearly talk! And, if these Pavlovian terms are left without neuro-physiological localization and foundation, the layman would be fully entitled to regard them as clinical descriptions analogous to other systems of psychology.

It would seem that the spheres of human response could have been argued in their own right as showing similarities—and then the theme of increasing tension leading to psychic and physiological débacle could have been demonstrated as between the physically and emotionally exhausted soldier, the religiously aroused ordinary decent man, and the victim of the terror, under all forms of mental and many forms of physical stress. In the text, the Pavlovian terms at times obscure the theme.

That Dr. Sargant invites the reader to abstract from the human experience those evidences of varying physiological stress and response, and to compare these, not only in different fields of human experience, but with evidence from non-human spheres, is in the best traditions of experimental medicine. The way in which he does this is in the tradition of provocation. To have reversed his order and led in the dog last, would have robbed the book of its sting, and left it only with its poignancy and gravity.

Dr. Sargant is to be thanked for his canine counter-irritant, and for producing a stimulating, and trenchant commentary on some affairs of all time and of now.

JONATHAN GOULD.

The Psychology of Sex Offenders. By A. ELLIS, Ph.D., and R. BRANCALE, M.D. Thomas, Springfield, Illinois; Blackwells, Oxford. 1956.

This reviews the first 300 cases referred to the New Jersey Diagnostic Centre, claiming to be the first "100 per cent. sample" of convicted sex-offenders. The authors have sadly missed an excellent opportunity for clinical reporting, but give some interesting information despite their obvious endeavour to justify the existence and preservation of their diagnostic centre, and to encourage its reduplication throughout the U.S.A. The literature on sex-offenders is almost entirely ignored. Perhaps the most interesting clinical information to be derived from this book is that the authors, a psychologist and a psychiatrist, consider 14 per cent. of their cases to be normal, and 29 per cent. to suffer from mild neurosis, but that 91 per cent. were "seriously

emotionally immature", and they opine that it is probably true of the vast majority of American adults! Forensically, they support:

1. Conviction by court of law.
2. Commitment to treatment as out-patient or in-patient by the court on the instruction of the psychologist/psychiatrist—p.s.w. team, and
3. Determination of the indeterminate commitment at the discretion of the psychiatrist in later charge of the case.

They give no thought at all to the possibility of difference of subsequent professional opinion regarding the suitability of the initial recommendations, and would place the diagnostic centre's team above the law, and the psychiatrist responsible for continued treatment, as far as resolution of any doubt at any stage of progress may be concerned.

Some of their main recommendations for the prevention of sex-offences are (1) to reduce drastically the number of sex-offences on the statute book; (2) to increase the amount and objectivity of sex instruction; (3) to "encourage a much more liberal, socially sanctioned heterosexual participation on the part of young people".

There are also appended a couple of reprints of older articles by the psychological author.

JONATHAN GOULD.

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Frontal Lobotomy 1936-1956

Three thousand lobotomy patients have been compared in 3 different categories: (1) Prefrontal lobotomy versus transorbital lobotomy; (2) private patients versus state hospital patients; (3) according to personality reaction type, schizophrenic, affective and psychoneurotic.

Follow-up studies reveal that following prefrontal lobotomy some 70 per cent. of schizophrenics, 80 per cent. of affectives, and 90 per cent. of psychoneurotics are functioning outside of the hospital in the 5- to 10-year period. This figure is twice as high in private patients as it is in state hospital patients.

Transorbital lobotomy is safer, more effective (with the exception of the hallucinated schizophrenic patients), and far more applicable to the problem of the state hospital, than is prefrontal lobotomy.

Multiple operations have been performed in about 1 patient in 10, with eventual satisfactory results in a third of them. When it is considered that this fraction amounts to 100 patients out of the hospital, this figure acquires significance.

Hospitals that select patients for operation with a view to release, and that encourage the relatives of patients to participate actively in the management of convalescence, enjoy a much higher percentage of released patients than do those that employ lobotomy more for the control of disturbed behaviour.

(Author's Abstr.)

Psychotomimetics, Clinical and Theoretical Considerations: Harmine, Win-2299 and Nalline

Harmine, Win-2299, and Nalline in single dosage produce many new mental effects in schizophrenics grossly similar to those elicited by mescaline and LSD. Many of the same effects are reported in normals after Harmine and Nalline (other workers). Unlike mescaline and LSD at usual dosage levels, the present psychotomimetics regularly produce drowsiness and sleep along with the aberrant mental effects. The resultant state is partly that of "hypnagogic" visual hallucinations or imagery. The results with increased dosage suggest that the basic effect of these agents is to produce an acute toxic reaction type. The difference between them and mescaline or LSD with respect to clouding of consciousness and certain aspects of the hallucinogenic response may be quantitative rather than qualitative. The indole nucleus is not necessary in the structure of psychotomimetics since Win-2299 and Nalline are non-indoles. The tertiary nitrogen grouping may contribute to certain aspects of psychotomimetic action.

(Authors' Abstr.)

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Studies on the Diethylamide of Lysergic Acid (LSD-25)

1. Chlorpromazine ameliorates partially the abnormal mental state induced by the diethylamide of lysergic acid (LSD-25) in man. Chlorpromazine has this effect when administered before or after LSD.

2. Azacyclonol (Frenquel) does not reduce the intensity of the LSD psychosis in man.

3. Reserpine does not mitigate the LSD psychosis in man. Patients receiving a combination of reserpine and LSD have severer symptoms than when receiving either drug alone. (Authors' Abstr.)

Studies on Phenylketonuria

Young phenylketonuric children frequently have seizures, usually in the form of infantile spasms. In that age group, hypsarhythmia and multiple seizure foci are found on electroencephalography. As these children grow older, they may have tonic-clonic convulsions, and their EEGs tend to show focal or generalized spike discharges. Of 23 patients, 7 had spike-and-

wave complexes that were similar to those found in petit mal epilepsy. Only one adult had normal EEGs.

EEG patterns vary with age regardless of etiology.

Eight patients undergoing treatment with phenylalanine-restricted diet had EEG examinations. Five of these showed an EEG improvement on the diet; two in this group showed increasingly abnormal EEGs when treatment was interrupted. In two other children no remarkable EEG changes could be noted. The eighth patient had only one EEG recorded.

These studies suggest that the phenylalanine-restricted diet has beneficial effect on seizures and EEGs of phenylketonuric children. A warning of caution against over-interpretation should be sounded, however, because EEGs will change with age, with stage of waking, drowsiness, and sleep, and only EEGs obtained under very similar conditions should be compared.

(Authors' Abstr.)

Conversion of Adrenaline to Adrenolutin in Human Blood Serum

Adrenochrome (3-hydroxy-N-methyl-5,6-dioxindole) and adrenolutin (3,5,6-trihydroxy-1-methylindole) may be involved in the production of schizophrenia. These compounds have not been detected in blood, nor have enzyme systems been clearly demonstrated which can produce them from adrenaline. It is therefore of interest to show that the conversion can occur in blood serum.

Following Osmond and Smythies' suggestion that schizophrenic patients may have within them an M substance related in structure to both mescaline and epinephrine, Hoffer, Osmond, and Smythies discovered that adrenochrome, an oxidized derivative of epinephrine, induced psychological changes in humans. Hoffer and Osmond postulated that the basic physiological abnormality in schizophrenia was an abnormality in the autonomic nervous system expressed chemically in the increased production of both acetylcholine and some oxidized derivative of adrenaline similar in structure to either adrenochrome or adrenolutin. Both these substances have similar properties in producing psychological changes.

The presence of both adrenochrome and adrenolutin in human blood serum has not been demonstrated, although enzyme systems that could achieve this conversion are known. Recently, Leach and Heath demonstrated that epinephrine added to schizophrenic serum was rapidly converted to a new substance having an absorption peak at 395 m μ . To a less degree the same conversion occurred in normal blood. The degree of conversion was significantly greater in schizophrenic serum than in normal serum. The conversion was accelerated by the addition of copper ions and inhibited by the addition of cyanide. They therefore believe that the enzyme is a copper-containing phenolase, similar to tyrosinase. When adrenochrome was added to serum, it was even more quickly converted to the new substance.

Adrenolutin, a reduced derivative of adrenochrome, might be more stable in a reducing medium such as serum, which contains ascorbic acid, glutathione, and proteins rich in sulfhydryl groups. Adrenolutin was therefore added to serum following in detail the procedure of Leach and Heath.

The absorption curve obtained with adrenolutin after 1 minute is similar to the one obtained by incubating epinephrine in schizophrenic blood for 80 minutes. After 80 minutes there is little change in the curve. This suggests that the substance formed in blood serum from adrenaline is adrenolutin.

In conclusion, it has been found that the enzyme which, according to Leach and Heath, occurs in higher concentration in schizophrenic than in normal blood, converts adrenaline to adrenolutin. This reinforces the suggestion that this oxidized derivative may play a role in the production of schizophrenia.

(Authors' Abstr.)

Lysergic Acid Diethylamide (LSD-25) Antagonists

Crude brain extract up to concentrations of 2 mg. per cubic centimeter of the mixture does not essentially change the behaviour of the Siamese fighting fish.

If the fish are allowed to remain in contact with this concentration of crude brain extract for two hours, addition of LSD-25 (2 γ per cubic centimeter) does not have its usual effect.

Whereas 0.02 and 0.2 mg. of crude brain extract per cubic centimeter have a minor effect on the LSD reaction, 2 mg. of crude brain extract per cubic centimeter blocks the LSD reaction. The LSD reaction observed corresponds to a concentration of 0.2 γ of LSD per cubic centimeter.

If the blocking effect occurs within the fish, this is apparently the first time that blocking action (not a symptomatic depressant) of behaviour changes engendered by LSD has been produced by a biologically derived substance.

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A Working Hypothesis as to the Nature of Hypnosis

"Animal hypnosis" (Totstellreflex), which represents one of the basic types of reactions common to man and animals, was used as an experimental pathogenetic model for the study of the mechanism of several psychiatric syndromes (catatonia, hypnoid syndrome, etc.). The course of this reaction in phylogenesis and ontogenesis was studied, and an EEG analysis was made in the rabbit.

"Animal hypnosis" is paroxysmally initiated central inhibition, which originates in the subcortical regions of the brain and from there spreads to the cerebral hemispheres. The termination of this state is realized by the ascending reticular activating system (Magoun), the function of which is not impaired in the course of "animal hypnosis".

The susceptibility of this paroxysmal inhibition to react to certain stimuli decreases in the course of ontogenesis. The same applies to the phylogenetic development of vertebrates with the neocortex. It follows that the old mechanism of "animal hypnosis", which is localized in the brain stem, is dominated during evolution more and more by younger cortical mechanisms, which progressively displace the reaction of paroxysmal inhibition from the normal animal's behaviour and finally lead to its disappearance.

The opinion is expressed that the reaction of "animal hypnosis"—paroxysmal inhibition—appears in higher evolutionary forms only in the case that normal subordination interrelations of individual parts of the brain are disturbed to such an extent that old, preformed mechanisms of the brain stem, which are inhibited under normal conditions by younger functional structures, come into play or become manifest. Such a situation occurs only under pathological conditions, and that is the reason that "animal hypnosis" in higher evolutionary forms and in man must be considered to be a pathological reaction, an instinctive, phylogenetically preformed mechanism, which appears in regressive forms of behaviour.

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The Incidence of Ammon's Horn Sclerosis

The findings of the present investigation and the interpretation placed upon them may be summarized as follows:

1. Out of 65 cases, selected only to avoid those patients with chronic organic disease of the brain, 5 (8 per cent.) showed lesions of the Ammon's horns all of which were of recent onset. These were considered in each case to be related to terminal disease or to the development of severe hypoxia in the hours or days before death. In none of the 65 cases was Ammon's horn sclerosis found, and there was no evidence to suggest that this could be looked on as a common (or indeed as an uncommon) incidental finding.

2. It was again confirmed that of those cases with organic disease of the brain the incidence of Ammon's horn lesions was essentially confined to the arteriosclerotic and the senile groups (including Alzheimer's disease) and to cases of general paresis, although very few cases of this were examined.

3. The incidence of lesions in the group of epileptic patients was roughly the same as that in the senile and the arteriosclerotic groups, but differences both in the morphological appearances of the lesions and in the mechanism of their production, in so far as this is known, suggest that such lesions should not be grouped together indiscriminately. In the senile and arteriosclerotic cases the development of the lesion occurs during the last few years of life and is found in the presence of severe, widespread degenerative changes occurring throughout the brain.

In the senile these seem to be part of a generalized disintegration of cerebral tissue (although there is no explanation why this should be so strikingly intense in the hippocampus). In the arteriosclerotic the lesion is that of infarction related to vascular disease. Since, how-

ever, vascular change plays a part in the senile process no single explanation is likely to be adequate.

4. In the adult cases of cryptogenic epilepsy the appearances are constantly those of a lesion of some considerable duration, and the evidence supports the view that this originates as the result of anoxia occurring in the early years of life, either as a birth injury or as the result of some upset in early childhood. Since convulsions, however, may themselves result in severe hypoxia they alone may account for the presence of a lesion.

(Author's Abstr.)

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Procedures Changing the Carbonic Anhydrase Activity of the Central Nervous System
The results of attempts to change the carbonic anhydrase content of the brain are given. Zinc salts, when given either parenterally or orally, failed to increase carbonic anhydrase in the brain of adult rats, nurslings or of the young mothers treated during pregnancy. Alcohol feedings did not decrease carbonic anhydrase in rat brain. Attempts to increase carbonic anhydrase in the brains of growing rats with carbon dioxide were negative except in one instance in which normal growth occurred during treatment. Forced blood production by simulated altitude resulted in increases in carbonic anhydrase in the cerebellum and cerebrum of rats after a 65 day exposure. Bone marrow caused a rostral progression of increase in carbonic anhydrase in growing rats in which the brain stem, cerebellum and cerebrum were tested. Results similar to those with bone marrow were found with the use of chlorpromazine. Large increases in carbonic anhydrase eventually occurred in the kidney with injections of both bone marrow and chlorpromazine. Electric shock in guinea pigs reduced the carbonic anhydrase in those parts of the brain more directly between the electrodes. The possible significance of the positive findings is discussed.

(Authors' Abstr.)

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The Entorhinal Area; Electrophysiological Studies of its Interrelations with Rhinencephalic Structures and the Brainstem

1. Electrophysiological investigations have been performed in the rhinencephalon of 16 specimens of the marsupial phalanger (*Trichosurus vulpecula*).
2. The reciprocity of pathways between entorhinal area, hippocampus and fornix was tested. It was found that whereas stimulation of the fornix was followed by responses in both the hippocampus and entorhinal area, stimulation of the entorhinal area evoked responses in the hippocampus, but only small irregular responses in the fornix. The findings on entorhinal stimulation thus throw some doubt on the classical concepts of Papez (1937) of major projections from entorhinal area to fornix through the hippocampal formation.

3. Interaction patterns were studied in the hippocampal responses following paired stimuli to fornix and entorhinal area. It was found that a conditioning stimulus to the fornix inhibited the test response to entorhinal stimulation. Conversely, a conditioning stimulus to the entorhinal area was followed by facilitation of the test response to fornix stimulation at many shock spacings. A "turnover" point in the hippocampal response to fornix stimulation was seen in the vicinity of the dentate pyramidal cells during progressive penetration of the hippocampal formation.

4. Interaction patterns were studied in the entorhinal area to paired shocks to widely separated points in the dorsal and ventral regions of the hippocampus. Significant differences were found in the extent of projections between these two parts of the hippocampus and the entorhinal area. Stimulation of the dorsal hippocampus was followed by large recruiting responses in the whole length of the entorhinal area. Stimulation of the ventral hippocampus was followed by entorhinal responses with much less evidence of recruitment. Stimulation of the entorhinal area was followed by smaller responses in the ventral hippocampus than in the dorsal, with fewer slow-wave trains in the ventral hippocampal responses.

5. Interaction studies suggest that an influx from the dorsal hippocampus can exercise a powerful influence over activity in the whole length of the entorhinal area. Although ventral areas of hippocampus do not appear similarly potent, the evidence suggests the presence of large projections between ventral and dorsal regions of the hippocampus through which the ventral hippocampus may exert its influence on patterns of intra-hippocampal integration.

6. Efferent projections from the entorhinal area to the midbrain tegmentum have been examined. The findings confirm previous histological studies (Adey, Merrillees and Sunderland, 1956) that a bilateral efferent pathway exists through the stria medullaris from the entorhinal area. Responses in it from entorhinal stimulation have many of the characteristics of a monosynaptic pathway, and exhibit a significantly shorter latency than those induced in the stria from hippocampal stimulation. The latency differences are compatible with the concept of a hippocampal projection to the stria medullaris through the entorhinal area. Responses in the midbrain at the intercollicular level were localized to the dorsal tegmentum in a region adjoining the periventricular gray matter where previous histological studies had suggested that fibers from the entorhinal area might terminate.

(Authors' Abstr.)

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The Effect of Phenylephrine, Methamphetamine, Cocaine, and Serotonin Upon the Adrenaline-Sensitive Component of the Reticular Activating System

A series of sympathomimetic agents and related substances were tested for their effect upon the EEG and blood pressure of the unanesthetized cat with mid-reticular coagulation. Adrenaline, noradrenaline, and phenylephrine all produced prompt, short-lasting and reproducible EEG activation and rise in blood pressure, but showed certain quantitative differences. Methamphetamine produced a feeble direct effect upon the EEG and blood pressure, and both responses showed tachyphylaxis. Both methamphetamine and cocaine were able to lower the threshold to adrenaline EEG activation ten-fold or more, and in large enough cumulative doses produced a sustained EEG activation which was abolished by further destruction of the mesencephalic tegmentum. Serotonin produced alternate phases of EEG activation and deactivation dissimilar in pattern to that produced by the sympathomimetic agents.

It is postulated that the central nervous system stimulating properties of methamphetamine and cocaine observed clinically are due to their sensitizing effect upon an adrenergic component of the reticular activating system.

(Author's Abstr.)

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Studies on Hallucinogenic Snuffs. Physiologically Active Components

Examination of seeds of *Piptadenia peregrina* obtained from Puerto Rico showed that the principal alkaloid present was bufotenine, an indole compound closely related to serotonin. Bufotenine had never before been isolated from a plant source although it has been found in the skin glands of toads and in certain mushrooms. Later on, two samples of snuff, one from Venezuela and one from Colombia, were studied chemically; and, again, bufotenine was found to be present in large quantities. Since the seeds of *Piptadenia* sp. are the only presently known seed source of bufotenine, strong support for the claims of Safford was thus established. Paper chromatographic comparisons of extracts of the snuffs and seeds provided the analytical evidence.

Further chemical study of various seed extracts resulted in the isolation of additional organic bases closely related to bufotenine. One such source *Piptadenia macrocarpa* contained five related indole compounds: bufotenine, N,N-dimethyltryptamine, bufotenine oxide, N,N-dimethyltryptamine oxide, and a substance of unknown structure. Structural comparisons of these materials with serotonin, the powerful vasoconstrictor which has received much attention recently, and with tryptophan from which all are undoubtedly derived, bring up interesting pharmacological questions: What physiological action, if any, do these compounds have? How do the slight variations in structure affect activity? Work done at the National Heart Institute and elsewhere has indicated that bufotenine, N,N-dimethyltryptamine and serotonin all have grossly similar effects on the cardiovascular system of the dog and cat. No obvious central nervous system effects were observed in these animals at a low dose level. The oxides were much less active than the bases. In the studies of Dr. Edward Evarts of the National Institute of Mental Health, it was found that the effects of bufotenine in the monkey were very similar to those of LSD, and that bufotenine and N,N-dimethyltryptamine exhibited similar actions on the synaptic junction of the optic nerve of the cat at approximately equivalent dose levels.

It should be pointed out that pharmacological examination of other fractions of seed extracts has not, as yet, afforded any evidence that physiologically active agents other than the five indole compounds are present. This possibility must not be excluded, however, when one considers the frequent observations pertaining to species differences with respect to physiological activity, along with the fact that animals are not ideal subjects for the study of drug-produced psychoses. For example, in man, LSD is active at a dose level of 0.0003

mg./kg. In the monkey, doses of 1 mg./kg. of LSD and 3 mg./kg. of bufotenine are employed for producing profound behavioral disturbances. This difference is 10,000-fold.

Chemical, Enzymatic and Metabolic Studies

A great deal of work on the chemistry of these compounds has been in progress. It will be sufficient here to state only that some of these chemical studies were directed toward proof of structure of the indole components of the seeds and that others were directed, as closely as possible, to the duplication of enzymatic conditions. It was found in enzymatic work that some of the products obtained were identical with those previously isolated from chemical reactions. For example, the soluble and microsomal fraction of mouse liver homogenate converts N,N-dimethyltryptamine to its oxide. When the oxide is incubated with whole homogenate, some of it is reduced back to the parent amine. These same reactions were also carried out under mild chemical conditions. The mitochondrial fraction of mouse liver homogenate converts N,N-dimethyltryptamine to 8-indoleacetic acid. This is not an unexpected result when one considers the metabolism of bufotenine in man. When Dr. H. D. Fabing administered bufotenine (the 5-hydroxy analog of N,N-dimethyltryptamine) to convicts at the Ohio State Penitentiary, urine samples were collected and examined. It was found that a small fraction of the bufotenine was excreted unchanged, but the major portion was converted to and excreted as 5-hydroxy-3-indoleacetic acid. Since, as stated above, oxides of tertiary amines are formed by mammalian enzymes, the next step was to learn if these oxides would undergo an enzymatic reaction. Results with the oxides of bufotenine, and N,N-dimethyltryptamine have, so far, been negative, but it was found that the oxide of N,N-dimethyltryptophan, an amino acid oxide which was prepared synthetically, did undergo this type of reaction. One of the N-methyl groups was eliminated from the molecule. Another unidentified product was formed. In the latter case, carbon dioxide was lost from the molecule.

The results of these enzymatic experiments have led to the conclusion that a new sequence of tryptophan metabolism should be proposed for plants and possibly for mammalian organisms. The next step will be to learn if any of these compounds plays a vital role in normal or, perhaps, abnormal physiological processes.

(Authors' Abstr.)

An Examination of Phenothiazine Derivatives with Comparisons of their Effects on the Alerting Reaction, Chemical Structure and Therapeutic Efficacy

A series of phenothiazine derivatives was examined to determine the effect on the reticular formation. Three derivatives which failed to block the alerting reaction to pain were inferior to chlorpromazine in the management of disturbed psychotic patients. It is therefore suggested that failure to depress the alerting reaction may be used as a screening procedure of new phenothiazine derivatives. But the converse does not hold, and their ability to inhibit alerting is not an adequate sign of therapeutic efficacy. In general, phenothiazine derivatives with 3 carbons in a straight chain are more effective tranquilizers than others with a greater or lesser number of carbons.

(Authors' Abstr.)

Behaviour of the Salamander Under the Influence of LSD-25 and Frenquel and Accompanying Electrical Activity of Brain and Spinal Cord

1. In the salamander, LSD-25 depresses the behavioural responses of touch, righting, swimming, walking, and vision, but the corneal response remains. During the peak of drug action the salamander assumes statue-like postures, and the electrical activity of the spinal cord shows a sustained discharge of waves of high frequency and amplitude.

2. Frenquel modifies the behaviour of the salamander by producing unsteadiness of gait, weakness of forelimbs, and a wobbly method of swimming. During the height of Frenquel action the electrical activity of both brain and spinal cord shows waves of very low amplitude.

3. With the dosages and methods used in this investigation, Frenquel and LSD-25 in some measure mutually modify the effects of one another. When the drugs are used together, they exert a depressant effect on behavioural responses and produce a weakened condition.

(Authors' Abstr.)

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Variations in Consciousness Produced by Stimulating Reticular Formation of the Monkey
 Stimulation of various areas in the reticular formation of the macaca mulatta produced a change in behaviour which the authors infer is due to a change in the state of consciousness. They believe such a change in consciousness is not dissimilar to that seen in humans experiencing an epileptic episode of either the petit mal or a psychomotor variant type.
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Functional Relationships Between the Red Nucleus and the Brachium Conjunctivum. Physiologic Study of Lesions of the Red Nucleus in Monkeys with Degenerated Superior Cerebellar Brachia

An attempt was made to determine the physiologic effects of localized lesions in the red nucleus in monkeys with complete and partial degeneration of the superior cerebellar peduncles. In ten rhesus monkeys portions of the deep cerebellar nuclei were removed by suction, and in one additional animal the brachium conjunctivum was completely sectioned caudal to the inferior colliculus. After observing these animals for periods of approximately 200 days, attempts were made to produce secondary stereotaxic lesions in the red nuclei. The latter procedures were successful in five animals. Serial sections of the brains and portions of the spinal cords were prepared by the Marchi method.

The following conclusions were drawn:

1. Lesions of the red nuclei in the monkey produce marked hypokinesia and increase the degree of hypokinesia associated with lesions of the deep cerebellar nuclei and the brachium conjunctivum.
2. In monkeys with complete or virtually complete degeneration of the brachium conjunctivum, lesions of the red nuclei do not significantly alter ataxia, ataxic tremor, or asynergic disturbances.
3. Lesions of the red nuclei may provoke an increase in ataxia and a reappearance of ataxic and simple tremor in monkeys with partial degeneration of the brachium conjunctivum, particularly if fibers in the dorsomedial portion of this structure have been preserved.
4. Transient choreiform activity may result from localized lesions of the red nucleus, but only if portions of the brachium conjunctivum are under-generated.

The hypothesis is presented that dyskinetic phenomena resulting from localized lesions of the red nucleus in the monkey are a consequence of interruption of ascending fibers of the brachium conjunctivum rather than destruction of the cells in the red nucleus.

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